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## Original Communications

### STAB WOUND OF THE HEART
### ELECTROCARDIOGRAPHIC STUDIES OF TWO CASES*


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SUCCESSFUL suture of heart wounds is by no means rare. There have been over three hundred reported attempts with a mortality of 48 per cent. That the mortality is decreasing is shown by the statistics of Peck[1] (1909) who reported 161 cases with 63 per cent deaths, Pool[2] (1912) who added 79 instances with 47 per cent mortality, and Smith[3] (1923) who added 58 cases with a mortality of 33 per cent. When it is considered that these reports date only from the bold operations of Cappelen[4] (September, 1895), Farina[5] (March, 1896), and Rehm[6] (September, 1896), it is realized how much progress has been made in so short a time, and how we may look for surgical procedures upon the central circulatory system with the same confidence that we now view operations in the abdominal cavity.

The purpose of this paper is to report the successful suture of two cases of stab wound of the heart (Fig. 1) together with electrocardiographic and roentgenographic studies. The electrocardiograms are of interest since they show accurately the changes found in known anatomical lesions. The teleoroentgenograms likewise show changes following trauma of the epicardium and pericardium.

CASE 1.—J. H., No. 43226, a negro boy of eighteen was admitted to the Emory Division of Grady Hospital at 10:30 P.M., Jan. 8, 1931. A half-hour previously he had been stabbed in chest with a penknife. He ran about three blocks, collapsed, and was brought immediately to the hospital. His clothes were covered with blood and he was bleeding profusely from a wound in the fourth interspace two inches to the left of the sternal border. He was conscious but extremely restless,


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and begging for water. Both pupils were dilated, the extremities cold and clammy, and the radial pulse imperceptible. The heart sounds were weak, but could be counted ninety per minute. The rate soon dropped to sixty. The blood pressure could not be determined.

A diagnosis of cardiac tamponade from stab wound of the heart was made and immediate operation performed. While preparations for operation were being carried out, electrocardiograms were made (Fig. 2). Under procaine anesthesia a flap of skin and muscle from the second to the fifth left costal cartilages was

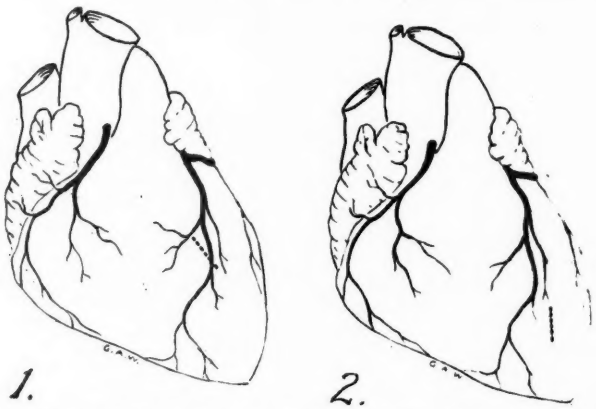


Fig. 1.—The approximate positions of the wounds are shown.

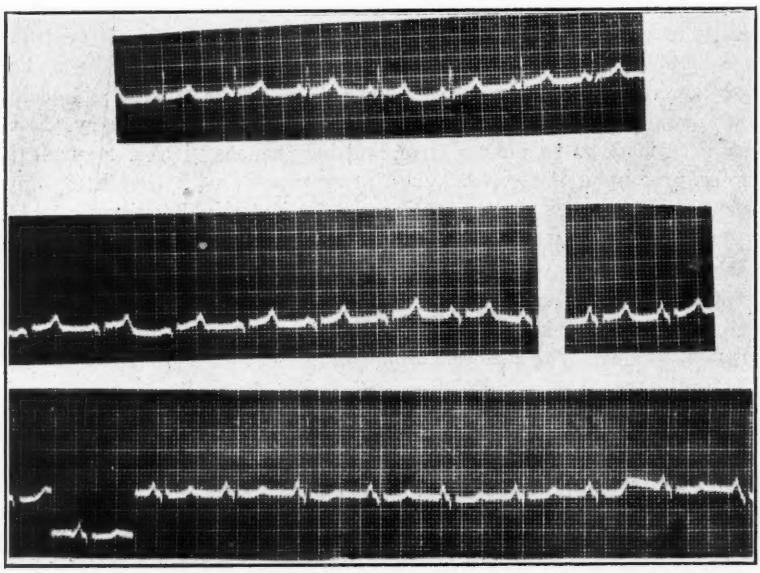

Fig. 2.—Case 1. Electrocardiogram made 40 minutes after stab wound severing descending branch of left coronary artery. Practically normal except for a changing form of P-wave, the significance of which is obscure.

Fig. 3.—Case 1. The patient 21 days after operation, showing type of incision and the point of wound.

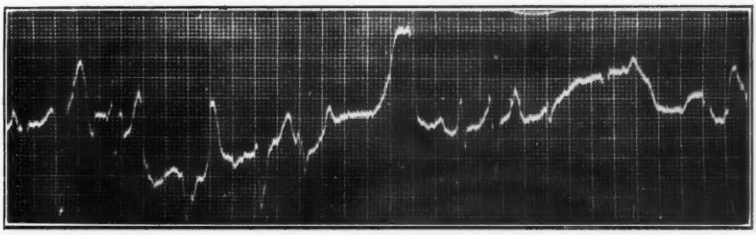

Fig. 4.—Case 1. Electrocardiogram made during operation. Lead II. The predominating type of complex is a ventricular premature beat, thought due to machanical stimulation produced by handling the heart. This closely simulates the record of a dying heart.

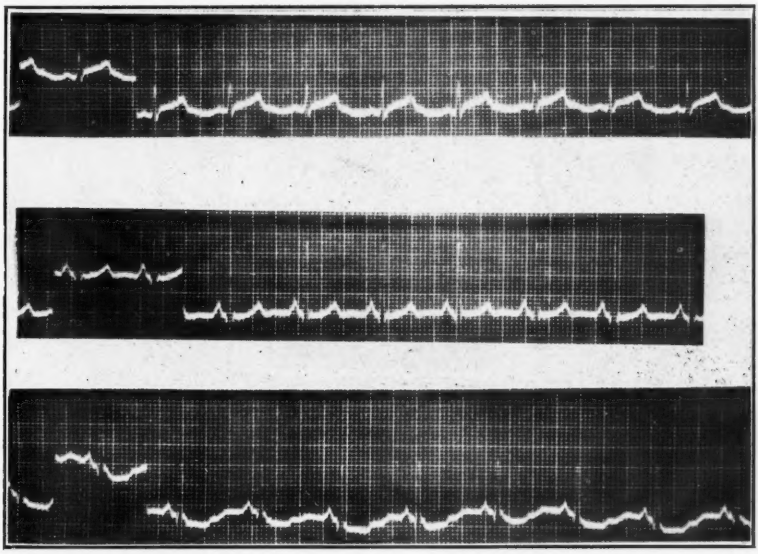

Fig. 5.—Case 1. Electrocardiogram made ten minutes after operation. Practically normal except for a slight elevation of the take-off of the T-wave in Lead I and a depression of the R-T interval in Lead III.

turned outward (Fig. 3). The third, fourth and fifth cartilages were removed, the internal mammary vessels tied, and the pericardium exposed by pushing back the overlying pleura and lung. A wound in the pericardium, which bled with each

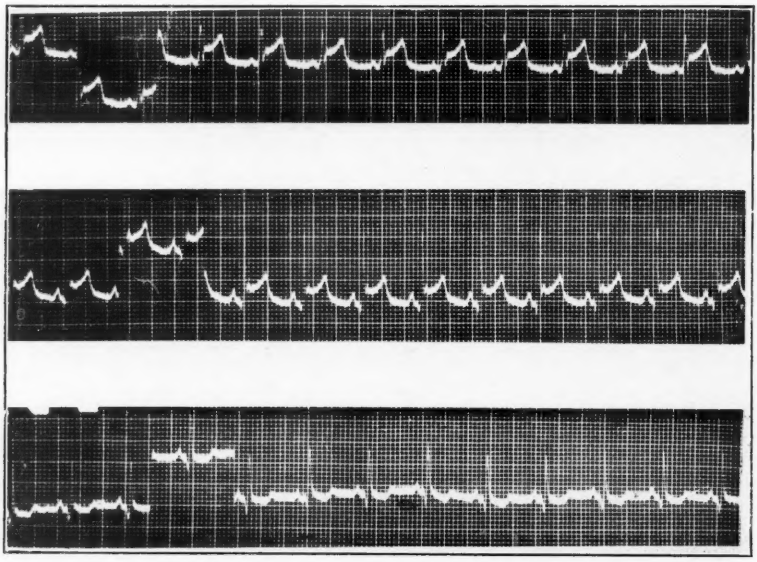

Fig. 6.—Case 1. Electrocardiogram thirty-six hours after operation, showing changes similar to those found in clinical coronary occulusion; viz., a marked elevation of the S-T interval in Leads I and II and a lesser elevation of the S-T interval with a biphasic T-wave in Lead III.

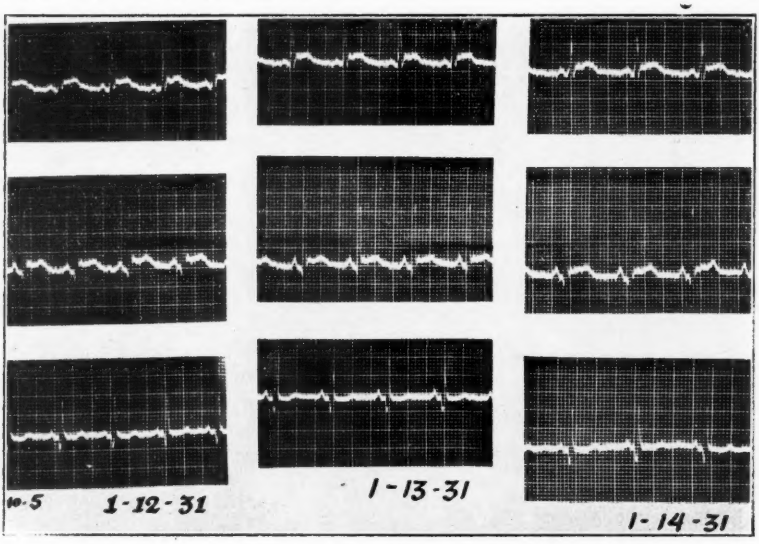


Fig. 7.—Case 1. Electrocardiograms made four, five and six days after operation. The tracing is similar to that seen in healing coronary occlusion. The S-T take-off is approaching the base line and the S-T interval is assuming a domed shape.

systole of the heart, was enlarged. The contractions of the heart were weak but regular. A finger was passed behind the heart and a large amount of fluid blood and clot evacuated. Following the removal of this, the heart began to beat strongly and the pulse became perceptible at the wrists. The heart wound, which was in the right ventricle just medial to the descending branch of the left coronary artery, began bleeding profusely. This was controlled by placing the left index finger on the wound and passing a suture of silk under the finger through the wound. This suture was then used both for hemostasis and steadying the heart while the wound was closed with two figure-of-eight sutures of silk. Electrocardiographic tracings were made during the suture (Fig. 4). After closure of the wound the descending branch of the left coronary artery and vein were found to be completely severed and the proximal and distal ends were ligated with silk.

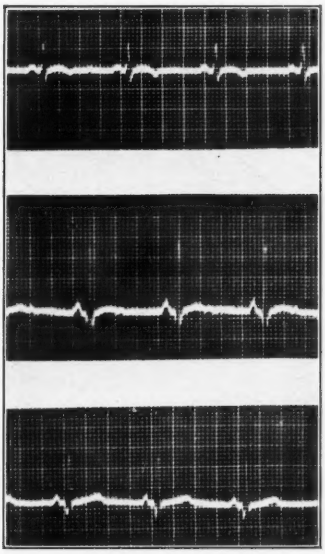

Fig. 8.

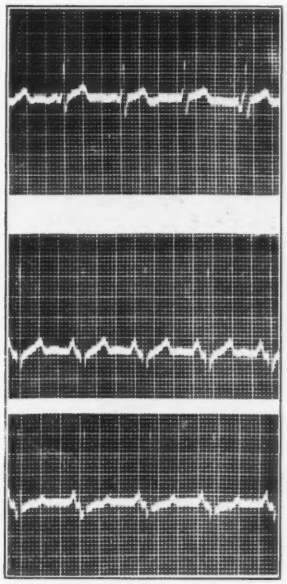

Fig. 9.

Fig. 8.—Case 1. Electrocardiogram sixteen days after operation. Evidence of further healing. The take-off of the S-T interval has reached the base line. The T-Wave is inverted in Lead I.

Fig. 9.—Case 1. Electrocardiogram three months after injury. Healing is practically complete. The T-wave is upright in all leads and the S-T interval takes off from the base line.

The pericardium was closed with interrupted sutures of silk, and the skin and muscle flap replaced and sutured with silkworm gut. A small drain of soft rubber was carried down to the pericardium. Forty-five minutes were required for the operation. Following closure of the skin an electrocardiographic tracing was made (Fig. 5), and this was repeated at frequent intervals (Figs. 6, 7, 8, 9). The pulse at the end of the operation was 90, and the systolic blood pressure 90, diastolic 60.

*Postoperative Course.*—The patient's temperature ranged between 99° F. and 101° F. for eight days after operation, and the pulse between 100 and 115. On

the second day the systolic blood pressure was 110 mm. of mercury and ranged between this and 120 mm. during his stay in the hospital.

On the third day there was a definite to and fro pericardial friction rub which persisted until the sixth day. There was considerable drainage and on removal of the drain on the fourth day, a large amount of fluid followed. A teleoroentgenogram made on the fourth day showed evidence of pericardial effusion and pneumonia of the left lower lung (Fig. 10). The teleoroentgenogram made twenty days after operation was normal (Fig. 11). The patient was allowed out of bed on the twentieth day and discharged from the hospital on the twenty-sixth day after operation. At present (June 1, 1931) he is symptom-free and apparently completely recovered.

## COMMENT

This case shows electrocardiographic evidence of damage to the anterior descending branch of the left coronary artery in man under

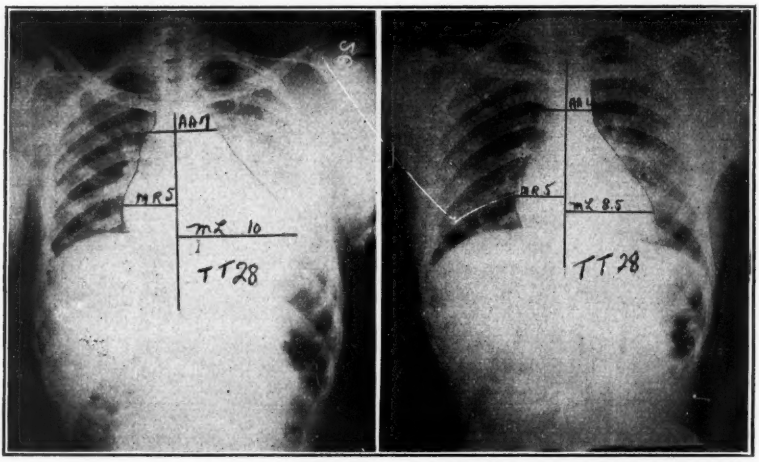


Fig. 10. Fig. 11.

Fig. 10.—Case 1. Teleoroentgenogram made four days after operation. There is definite evidence of pericardial effusion.

Fig. 11.—Case 1. Teleoroentgenogram made twenty days after operation. The cardiac measurements are normal.

experimental conditions. When the changes found in this patient are compared to those that occur in clinical occlusion of the same artery, certain differences are seen. Instead of the classical opposite deflections of the S-T interval in Leads I and III as shown by Barnes and Whitten,[7] the S-T interval deflection in this case is in the same direction in all three leads. A consideration of the work of Scott *et al.*[8] and Feil *et al.*[9] will probably explain these differences. They have shown that experimental ligation of the anterior descending branch of the left coronary artery may produce varying changes in the directional deflection of the S-T interval. They have also shown that peri-

cardial effusion may cause similar changes. This patient not only had pericardial effusion but also a normal myocardium and probably an abundant coronary anastomosis. Thus there is no reason that the electrocardiogram should exactly simulate that found in individuals with coronary and myocardial disease.

Fig. 12. Fig. 13.

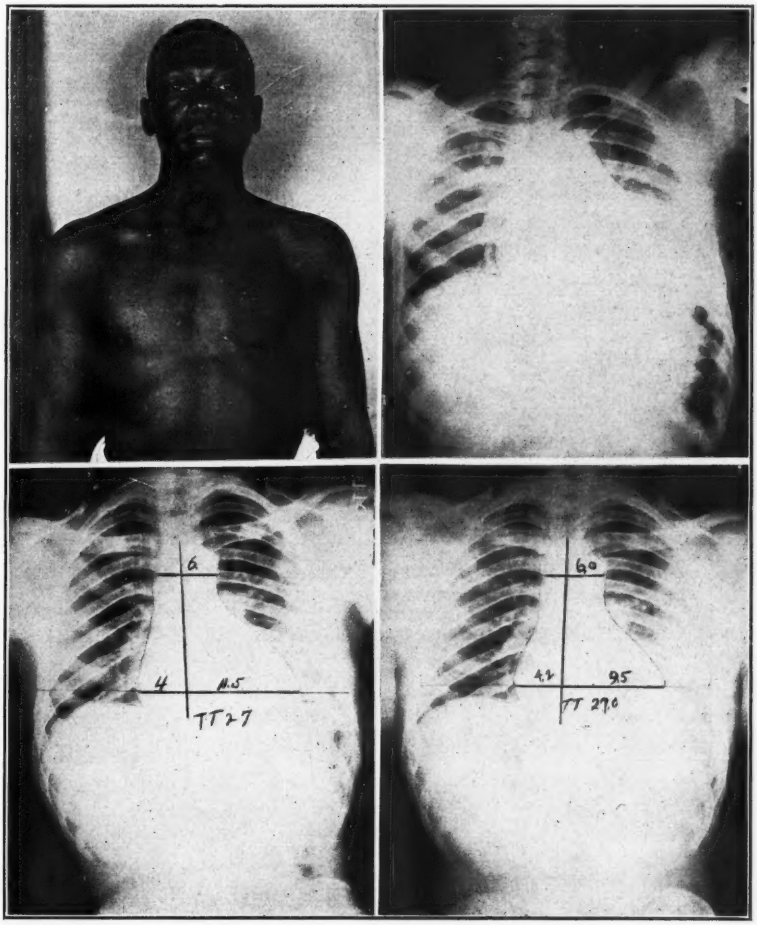


Fig. 14. Fig. 15.

Fig. 12.—Case 2. The patient twenty days after operation showing incision over the fifth costal cartilage.

Fig. 13.—Case 2. Roentgenogram four days after accident. Pericardial effusion and pneumonia of the left lower lung.

Fig. 14.—Case 2. Teleoroentgenogram twenty days after operation. Evidence of pericardial effusion and resolving pneumonia of the left lower lung.

Fig. 15.—Case 2. Teleoroentgenogram forty-five days after operation. Cardiac measurements normal.

It is of the greatest clinical importance that electrocardiographic evidence of coronary occlusion was not seen until thirty-six hours after the accident. This is in accord with the previously demonstrated observations of Levine[10] and is of extreme importance in differentiating an acute coronary occlusion and acute surgical diseases of the abdomen, notably ruptured peptic ulcer and gall bladder disease.

CASE 2.—T. M., No. 41739, a negro man of twenty-five was admitted to the Emory Division of Grady Hospital, Sept. 25, 1930. He had been stabbed in the chest with a knife about 40 minutes previously. He was bleeding freely from a

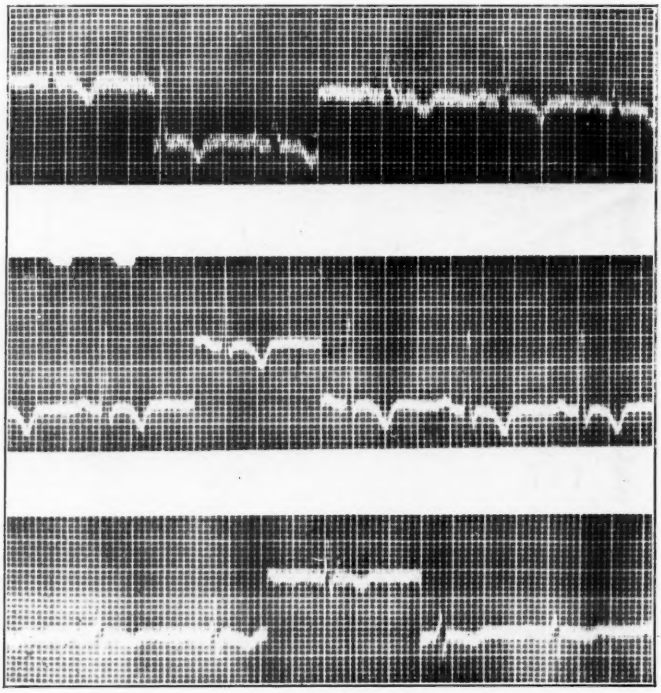

Fig. 16.—Case 2. Electrocardiogram made three weeks after operation. Evidence of myocardial healing. T-wave sharply inverted in Leads I and II with slightly dome-shaped S-T interval.

wound in the left fifth interspace near the sternal margin. He was rational, but weak and thirsty. The arterial blood pressure was 80 mm. of mercury, pulse 110, regular, but weak.

Under procaine anesthesia the skin and muscles over the fourth costal cartilage were incised and about three inches of this cartilage removed (Fig. 12). The pleura and lung were pushed laterally and the pericardium exposed. A wound in the pericardium, about an inch long, was bleeding profusely. This was enlarged and a wound 1.5 cm. long was discovered in the left ventricle just to the left of the descending branch of the left coronary artery. It was bleeding with each systole of the heart but was easily closed with two figure-of-eight sutures of

silk. The clot and free blood in the pericardium were then removed, and the cardiac contractions immediately became strong and regular. The pericardial opening was closed with a continuous suture of silk and the muscle and skin layers were closed without drainage with catgut and silk.

*Postoperative Course.*—The patient left the operating table in good condition. His pulse was 110, systolic blood pressure 90 mm. of mercury, and his respirations 30. An hour later he was given a transfusion of 500 c.c. of blood. His temperature ranged between 99° F. and 102° F. for five days after operation. Four days after

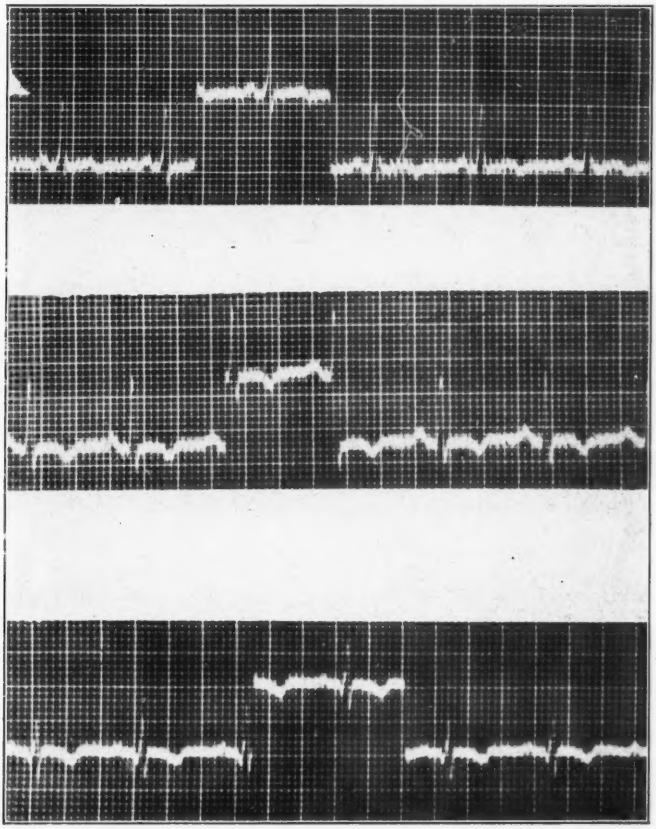

Fig. 17.—Case 2. Electrocardiogram seven weeks after operation. Evidence of further healing is shown by a decrease in the negativity of the T-wave in Leads I and II.

the accident there was evidence of cardiac enlargement and of pneumonia in the left lower lung; this was confirmed by a roentgenogram of the chest (Fig. 13). The heart sounds were diminished but regular in rhythm, the pulse 100, and the systolic blood pressure 115, diastolic 75. The cardiac enlargement was characteristic of pericardial effusion, but since there was no embarrassment of the circulation the fluid was not withdrawn. On Oct. 15, 1930 (20 days after the accident), there was still evidence of pericardial effusion both by physical signs and

by roentgenogram (Fig. 14), but there had been a definite decrease in the size of the cardiac shadow. The chest wound healed per primum and the patient was discharged from the hospital 26 days after operation. Forty-five days after operation the teleoroentgenogram was normal (Fig. 15) and at the present time (June 1, 1931) he is in good condition without any evidence of cardiac damage.

It is unfortunate that electrocardiograms were not made earlier in this case. The record made three weeks after the accident (Fig. 16), however, shows definite

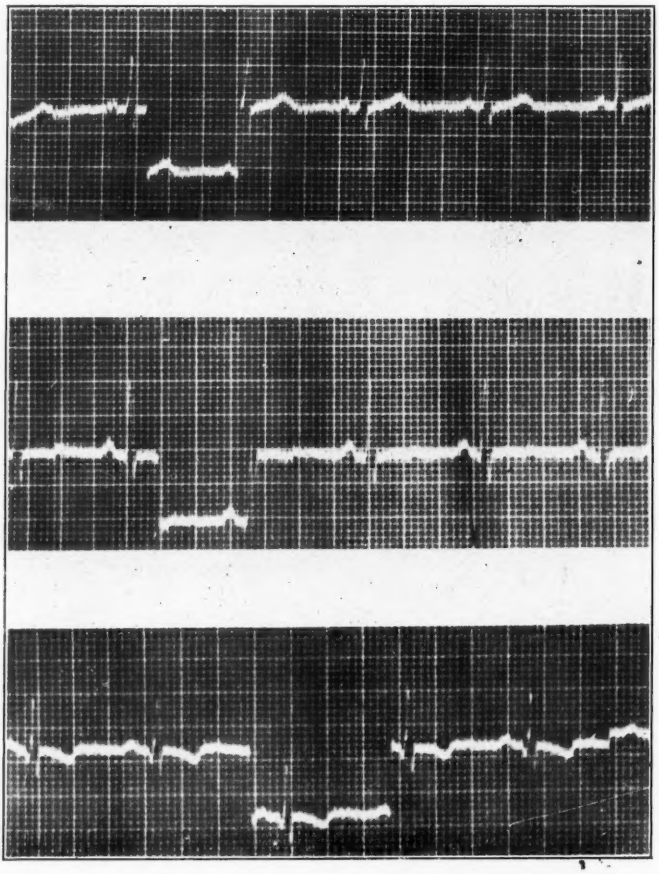

Fig. 18.—Case 2. Electrocardiogram made six months after the accident. The T-wave in Lead I is upright, in Lead II biphasic, and in Lead III inverted. The take-off of the S-T interval is normal.

evidence of myocardial damage seen in the healing of clinical coronary occlusion; viz., a sharply inverted T-wave in Leads I and II with a slightly dome shaped S-T interval. It is of interest that the electrocardiographic changes seen here were identical with those seen in the healing of clinical coronary occlusion although the vessels were not injured. The changes were therefore due merely to myocardial trauma which occurred near the anterior descending branch of the left coronary artery (Figs. 17 and 18).

## COMMENT

In this patient the wound was sutured before removal of the blood in the pericardium. This made suture easier since the heart was beating weakly as a result of the tamponade. It would seem that this is advisable since a suture can be quickly and easily passed and be used both for hemostasis and traction. As in Case 1, pericardial effusion occurred but was rapidly absorbed. Drainage with the added danger of infection would therefore seem unnecessary. Even though the coronary vessel was uninjured there were electrocardiographic changes similar to those found in coronary occlusion.

## CONCLUSIONS

1. Two cases of successful suture of stab wound of the heart are presented.

2. In one patient the wound was in the right ventricle with severance of the descending branch of the left coronary artery. In the other the wound was in the left ventricle and the coronary artery was uninjured.

3. The electrocardiographic findings in both cases were similar to those seen in clinical occlusion of the coronary arteries.

4. In the patient with ligation of the coronary artery, electrocardiographic changes did not appear until thirty-six hours after injury, a fact of clinical importance in differentiating between coronary occlusion and abdominal disease.

NOTE.—Electrocardiograms were made by Dr. W. K. Purks. Further electrocardiographic findings will be published by him elsewhere.

## REFERENCES

1. Peck, G. H.: The Operative Treatment of Heart Wounds, Ann. Surg. 50: 101, 1909.
2. Pool, E. H.: Treatment of Heart Wounds, Ann. Surg. 55: 485, 1912.
3. Smith, Wm. Randolph: Cardiorrhaphy in Acute Injuries, Ann. Surg. 78: 696, 1923.
4. Cappelan, A.: Vulnis Cordis, Sutur af Hjertet, Norsk. Mag. f. Laegevidensk. 11: 285, 1896.
5. Farina: Centralbl. f. Chir. 23: 1224, 1896.
6. Rehn, L.: Ueber Penetrienrende Herzwunden und Herznaht, Arch. f. klin. Chir. 315: 1897.
7. Barnes, A. R., and Whitten, M. B.: Study of the R-T Interval in Myocardial Infarction, Am. Heart J. 5: 142, 1929.
8. Scott, R. W., Feil, H. S., and Katz, L. N.: The Electrocardiogram in Pericardial Effusion, Am. Heart J. 5: 68, 1929.
9. Feil, H. S., Katz, L. N., Moore, R. A., and Scott, R. M.: The Electrocardiographic Changes in Myocardial Ischemia, Am. Heart J. 6: 522, 1931.
10. Levine, S. A.: Coronary Occlusion; Its Various Clinical Features, Medicine 8: 245, 1929.

## DISCUSSION

The discussion of this paper will be found with that of Dr. Hudson's paper at the end of Dr. Hudson's paper, p. 150.

# PNEUMOCARDIAC TAMPONADE
## A STUDY OF THE EFFECTS OF ATMOSPHERIC PRESSURE, NEGATIVE PRESSURE AND POSITIVE PRESSURE UPON THE HEART*

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### INTRODUCTION

WHEN the pressure upon the heart and the great vessels at the base of the heart is changed from the usual negative pressure of the thoracic cavity to the pressure of the atmosphere, a definite alteration in the circulation takes place. This alteration in the circulation was measured by Beck and Cox[1] and consisted of: (1) A definite sustained rise in the venous pressure; (2) a fall of from 8 to 30 mm. of mercury in the arterial pressure (this decrease in pressure was sustained in one experiment, in others it was transient); (3) a decrease of from 15 to 30 per cent in the minute volume output of the heart. The term applied to these changes in the circulation brought about by exposure of the heart to the atmosphere was pneumocardiac tamponade. A clinical application of pneumocardiac tamponade was made by Beck and Cox in the citation of a case in which the patient possessed only a slight circulatory reserve and was unable to withstand exposure of the heart to the atmosphere.

The fact that the circulation was altered when the heart was exposed to atmospheric pressure was not an entirely new idea to surgeons working in the thorax. Those investigators who did the experimental work on pneumothorax at the beginning of the present century made the observation that the jugular veins became distended when the chest was opened. This elevation in the venous pressure was attributed to a partial obstruction in the venous filling of the heart. To overcome this effect on the heart and also to prevent collapse of the lung a negative chamber was devised and used by Sauerbruch.[2] Of the various circulatory changes that accompany pneumothorax, little more than this rise in venous pressure was known, and when it was realized that the lungs could be inflated simply by the use of positive intrapharyngeal pressure, the negative chamber, so thoughtfully conceived and constructed, was discarded. Actual measurements of pneumocardiac tamponade were not carried out. It would seem that there

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were two reasons for this blind spot in our knowledge of pneumothorax. One was the difficulty of separating the effect of atmospheric pressure in the pericardial cavity from its effect in the pleural cavity. To avoid the complication of collapse of the lung it was necessary to suture the pericardium to the thoracic wall as a preliminary step before opening the pericardial cavity to the atmosphere. The other reason was the fact that cardiac output determinations constitute the best index of cardiac function, and during that period when pneumothorax was intensively studied, cardiac output determinations were not commonly carried out.

Because of the important rôle that these measurements seem to play, not only in the surgery of the central circulatory system, but also in the surgery of the thorax in general, further investigations on this subject were carried out. The circulatory effects of variable gas pressures applied to the heart and to the great vessels at the base of the heart were determined, and these studies are the subject of this paper.

*Method.*—The experiments were carried out on dogs. Under ether anesthesia the pericardium was sutured to the left thoracic wall near the sternum over an area about 4 cm. in diameter. This preliminary operation made it possible later on to open the pericardial cavity to the atmosphere without opening the pleura. After the wound had healed the dog was given a preliminary dose of morphine and then was anesthetized with barbital sodium, 0.3 gm. per kilogram, and various determinations were carried out. Pressure determinations and cardiac output determinations were not done on the same dog. At the completion of the experiment the dog was killed by chloroform narcosis. The arterial pressure was determined by connecting a mercury manometer to a carotid artery. The venous pressure was determined by a water manometer connected to a cannula inserted into the superior vena cava through the jugular vein. Graphic records were made by a Marey tambour. Intrathoracic pressures were measured by a water manometer or recorded by a tambour connected to a metal cannula inserted into the thorax.

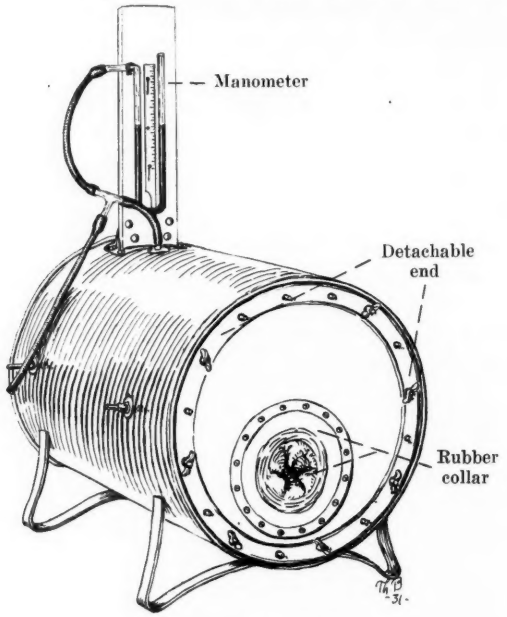
Cardiac output determinations were made by means of the Fick principle. The trachea was connected to a Benedict spirometer. Specimens of mixed venous blood were obtained from the right auricle by means of a metal cannula inserted into the right auricle through a jugular vein. After this cannula was inserted, it was not disturbed throughout the experiment. Its position in the right auricle was verified at necropsy. Arterial blood was obtained by tapping a carotid artery with a small aspirating needle. The artery was dissected from its bed and made accessible by anchoring sternocleidomastoid muscle beneath it to the trachea. The specimens of blood were drawn while the spirometer tracing was being made. The

specimens were examined immediately for $CO_2$ and $O_2$ content. The oxygen consumption was determined usually over a ten-minute period.

According to the Fick formula:

$$\frac{\text{Cubic centimeters of } O_2 \text{ consumed per minute}}{\text{Amount of } O_2 \text{ taken up by 1 c.c. of blood in passing through the lungs}} = \text{Cubic centimeters of blood passing through the lungs per minute or the cardiac output per minute}$$

The cardiac output determinations were a source of great labor and anxiety to us. The results fluctuated over such a wide range that most of them were of little value until we learned the importance of stabilizing the temperature of the dog. The temperature of the dog could be controlled over a five-hour period with a variation of less




Negative Pressure Chamber

Fig. 1.—The chamber used for the application of negative or positive pressure upon the heart. The end containing the rubber flange is detachable. The flange of rubber is slipped over the head and the body is inserted into the chamber. A measured amount of negative or positive pressure is applied to the chamber. This pressure is transmitted to the intrapericardial structures through the opening in the pericardium.

than one-half degree Centigrade. This was done by cooling the operating table with ice and by replacing the water in the spirometer with cold water so that the dog breathed chilled oxygen. The temperature was stabilized at 37° C. or 38° C., and when these precautions were taken the minute volume output determinations were usually acceptable. Occasionally for no apparent reason a wide deviation in result occurred. In such instances the determination was repeated or the experiment was discarded.

A metal chamber was constructed for the application of positive or negative pressure upon the heart (Fig. 1). One end of the chamber was detachable. This end contained a rubber diaphragm which could be slipped over the head of the dog. The body was then placed in the chamber and the demountable end was attached. The pericardial cavity was opened to the atmosphere before the dog was inserted into

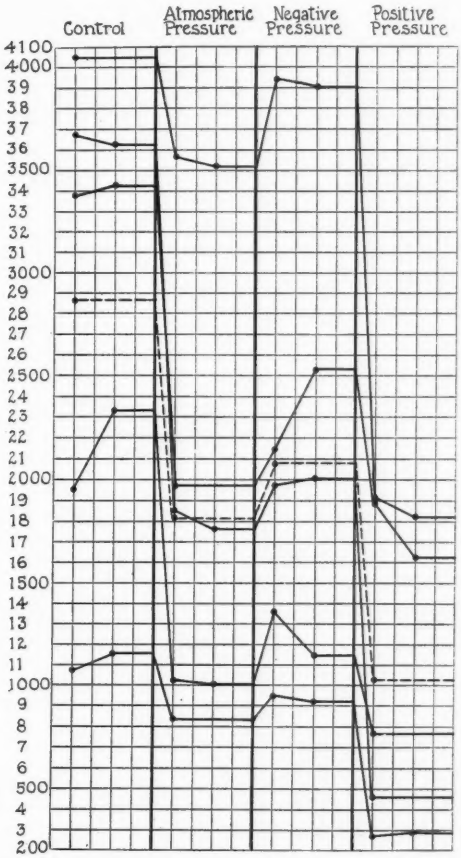



Fig. 2.—Minute volume output determinations in cubic centimeters in five consecutive experiments. When the pericardial cavity was opened to atmospheric pressure the cardiac output decreased. The application of negative pressure brought about partial recovery. Positive pressure applied to the intrapericardial structures brought about a marked fall in cardiac output. The broken line indicates the average of the five experiments.

the chamber. As the pressure in the chamber was varied, so also did the pressure vary upon the heart and the intrapericardial portions of the great vessels at the base of the heart. A manometer at the top of the chamber indicated the pressure within the chamber. An outlet

from the side was used to conduct intrapleural pressure. Ordinary water-traps were used to regulate the amount of pressure or suction applied within the chamber.

For most of the cardiac output determinations pressure or suction was applied to the intrapericardial structures simply through a catheter sutured hermetically into the opening in the thoracic wall. The chamber was used for pressure determinations.

### CARDIAC OUTPUT DETERMINATIONS

The five experiments recorded in Table I and Fig. 2 are those in which the temperature was regulated. An example of this type of experiment was as follows:

*Experiment 2.*—On May 14, 1931, a mongrel bulldog, weighing 15 kg., was anesthetized with ether. About 7 cm. of the left fifth rib was resected and the pericardium was securely sutured to the thoracic wall over an area about 4 cm. in diameter. The wound in the thoracic wall was sutured with silk.

On May 28, the dog was in good condition. The wound had healed. At 6:30 A.M., morphine, 0.15 gm., was given. At 7 A.M., 4.5 gm. of barbital sodium, mixed with about 200 c.c. of physiologic solution of sodium chloride was given intravenously. Complete surgical anesthesia appeared in a few minutes. The trachea was cannulated. The right carotid artery was elevated from its bed and made accessible by tacking the sternocleidomastoid muscle to the trachea beneath the artery. The left jugular vein was isolated and a cannula was inserted through the vein into the right auricle. The temperature of the dog was maintained throughout the experiment between 37.4° and 37.6° C. Cold water was placed in the spirometer.

At 9 A.M. an oxygen consumption curve was obtained over a ten-minute period. While this curve was being taken about 8 c.c. of blood was taken from the right auricle and then a similar amount was taken from the carotid artery under oil. Analyses were carried out immediately. The oxygen consumption per minute was 70.54 c.c. The oxygen content of the arterial blood was 20.63; of the venous blood, 17.03 volumes per cent. The arterial-venous difference was 3.60. The cardiac output per minute was 1,959 c.c.

At 9:30 A.M. this procedure was repeated. The oxygen consumption per minute was 69.26 c.c. The oxygen content of the arterial blood was 21.20, of the venous blood 18.22 volumes per cent. The arterial-venous difference was 2.98. The cardiac output per minute was 2,324 c.c.

After this oxygen consumption curve was obtained, an incision was made through the operative scar and an opening about 2 cm. in diameter was made into the pericardial cavity. As soon as this was done the pressure in the right auricle rose 3 cm. of water. The respiration increased slightly in rate and decreased slightly in depth.

At 10 o'clock the oxygen consumption per minute was 63.19 c.c. The oxygen content of the arterial blood was 23.13, of the venous blood 16.84 volumes per cent. The arterial-venous difference was 6.29. The cardiac output per minute was 1,004 c.c.

At 10:30 o'clock the oxygen consumption per minute was 69.64 c.c. The oxygen content of the arterial blood was 23.38; of the venous blood 16.42 volumes per cent. The arterial-venous difference was 6.96. The cardiac output per minute was 1,000 c.c.

A catheter was inserted through the opening into the pericardial cavity and suction equivalent to 7 cm. of water was applied. The right auricular pressure fell to about the normal level. The respiratory movements became slower and slightly deeper. About thirty minutes later a cardiac output determination was made. The oxygen consumption per minute was 67.33 c.c. The oxygen content of the arterial blood was 21.29; of the venous blood 16.34 volumes per cent. The arterial-venous difference was 4.95. The cardiac output per minute was 1,360 c.c.

At 11:30 another determination was carried out. The oxygen consumption per minute was 66.99 c.c. The oxygen content of the arterial blood was 22.01; of the venous blood 16:15 volumes per cent. The arterial-venous difference was 5.86. The cardiac output per minute was 1,143 c.c.

At 12 o'clock the pressure in the pericardial cavity was changed to 7 cm. of water above atmosphere. The right auricular pressure rose 7 or 8 cm. of water above the normal level. The respiratory movements became more rapid and more shallow. The oxygen consumption per minute was 68.17 c.c. The oxygen content of the arterial blood was 23.24; of the venous blood 14.51 volumes per cent. The arterial venous difference was 8.73. The cardiac output per minute was 780 c.c.

The dog was killed by chloroform narcosis. The end of the cannula inserted through the jugular vein lay in the right auricle. The opening into the pericardium did not communicate with the pleural cavity. The pericardium ballooned out when positive pressure was applied to the pericardium and it was apparent that this pressure exerted a compression effect upon the intrapericardial structures. Indeed, it actually forced blood to flow from the right auricle into the extrapericardial portions of the venae cavae. When suction was applied the pericardial cavity collapsed, and the parietal pericardium came into intimate contact with the surface of the heart.

The data on these cardiac output determinations are given in tabular form (Table I). The determinations were not always carried out in the same rotation as those recorded above. We learned that when the minute volume output was greatly decreased by positive pressure and then this pressure was replaced by suction that the output returned slowly over a period of hours. Therefore, we reserved the determination to show the effect of positive pressure as the last determination of the experiment.

In the following eight experiments the temperature of the dog was not controlled and the cardiac output determinations showed wide fluctuations due to shivering or panting. Furthermore, the determinations were not done in duplicate and therefore carry less significance than the results recorded above. The blood gas determinations are given in volumes per cent.

*Experiment 6.*—A mongrel, weighing 16 kg. A control determination was carried out. Arterial blood: $CO_2$ – 45.55, $O_2$ – 20.93; venous blood: $CO_2$ – 49.70, $O_2$ – 14.73. Oxygen consumption per minute was 120.37 c.c. The cardiac output per minute was 1,941 c.c.

The pericardial cavity was then opened to atmospheric pressure. Arterial blood: $CO_2$ – 46.52, $O_2$ – 20.95; venous blood: $CO_2$ – 50.30, $O_2$ – 14.24. Oxygen consumption per minute 92.64 c.c. Cardiac output per minute 1,380 c.c.

TABLE I

**Experiment 1. Police dog, weight 25 kg. Barbital sodium 7.5 gm. intravenously.**

| | Carbon dioxide content, per cent by volume, 1 c.c. of blood | | Oxygen content, per cent by volume, 1 c.c. of blood | | Arterial-venous difference | Oxygen consumption, c.c. per min. | Cardiac output, c.c. per min. |
|---|---|---|---|---|---|---|---|
| | Arterial | Venous | Arterial | Venous | | | |
| Control | 43.52 | 45.91 | 24.95 | 22.30 | 2.65 | 107.10 | 4041 |
| Pericardium opened to atmospheric pressure. Rt. auricular pressure | 44.04 | 46.56 | 25.28 | 22.37 | 2.91 | 103.98 | 3573 |
| rose 3.5 cm. water. | 45.33 | 47.12 | 24.05 | 21.15 | 2.90 | 101.71 | 3507 |
| Suction equivalent to 9 cm. water applied to pericardial cavity. Rt. auricular pressure fell | 48.04 | 49.48 | 23.11 | 20.44 | 2.67 | 105.36 | 3946 |
| to level of control. | 48.11 | 49.04 | 23.11 | 20.46 | 2.65 | 103.46 | 3900 |
| Positive pressure equivalent to 9 cm. water applied to pericardial cavity. Right auricular pressure rose to 10.5 cm. water | 44.45 | 47.32 | 27.35 | 22.44 | 4.91 | 93.35 | 1901 |
| above level of control. | 44.29 | 46.63 | 28.01 | 22.54 | 5.47 | 99.53 | 1819 |

**Experiment 2. Mongrel bulldog, weight 15 kg. Barbital sodium 4.5 gm. intravenously.**

| | Carbon dioxide content, per cent by volume, 1 c.c. of blood | | Oxygen content, per cent by volume, 1 c.c. of blood | | Arterial-venous difference | Oxygen consumption, c.c. per min. | Cardiac output, c.c. per min. |
|---|---|---|---|---|---|---|---|
| | Arterial | Venous | Arterial | Venous | | | |
| Control | 55.12 | 56.69 | 20.63 | 17.03 | 3.60 | 70.54 | 1959 |
| | 53.37 | 54.80 | 21.20 | 18.22 | 2.98 | 69.26 | 2324 |
| Pericardium opened to atmospheric pressure. Right auricular pressure | 44.98 | 50.05 | 23.13 | 16.84 | 6.29 | 63.19 | 1004 |
| rose 3 cm. water. | 44.17 | 48.22 | 23.38 | 16.42 | 6.96 | 69.64 | 1000 |
| Suction equivalent to 7 cm. water applied to pericardial cavity. Rt. auricular pressure fell approximately to | 45.67 | 49.08 | 21.29 | 16 34 | 4.95 | 67.33 | 1360 |
| level of control. | 44.95 | 48.37 | 22.01 | 16.15 | 5.86 | 66.99 | 1143 |
| Positive pressure equivalent to 7 cm. water applied to pericardial cavity. Rt. auricular pressure rose 8 cm. water above level of control. | 42.54 | 47.15 | 23.24 | 14.51 | 8.73 | 68.17 | 780 |

TABLE I—CONT'D

**Experiment 3. Mongrel, weight 17 kg. Barbital sodium 4.1 gm. intravenously.**

| | Carbon dioxide content, per cent by volume, 1 c.c. of blood | | Oxygen content, per cent by volume, 1 c.c. of blood | | Arterial-venous difference | Oxygen consumption, c.c. per min. | Cardiac output, c.c. per min. |
|---|---|---|---|---|---|---|---|
| | Arterial | Venous | Arterial | Venous | | | |
| Control | 53.65 | 53.99 | 21.09 | 18.79 | 2.30 | 84.50 | 3674 |
| | 53.43 | 54.38 | 21.73 | 19.48 | 2.25 | 81.73 | 3632 |
| Pericardium opened to atmospheric pressure. Rt. auricular pressure rose 2.5 cm. water. | 51.73 | 53.05 | 22.66 | 18.56 | 4.10 | 75.52 | 1842 |
| | 51.86 | 53.03 | 22.43 | 17.92 | 4.51 | 79.54 | 1763 |
| Suction equivalent to 8 cm. water applied to pericardial cavity. Right auricular pressure fell to level of control. | 52.51 | 54.08 | 21.28 | 17.66 | 3.62 | 71.34 | 1970 |
| | 50.94 | 54.41 | 21.02 | 17.36 | 3.66 | 73.51 | 2000 |
| Positive pressure equivalent to 8 cm. water applied to pericardial cavity. Rt. auricular pressure rose 11.5 cm. above level of control. | 44.39 | 51.73 | 25.23 | 12.33 | 12.90 | 63.67 | 458 |

**Experiment 4. Mongrel bulldog, weight 15 kg. Barbital sodium 4.5 gm. intravenously.**

| | Carbon dioxide content, per cent by volume, 1 c.c. of blood | | Oxygen content, per cent by volume, 1 c.c. of blood | | Arterial-venous difference | Oxygen consumption, c.c. per min. | Cardiac output, c.c. per min. |
|---|---|---|---|---|---|---|---|
| | Arterial | Venous | Arterial | Venous | | | |
| Control | 54.93 | 57.55 | 20.08 | 15.67 | 4.41 | 48.00 | 1088 |
| | 51.20 | 53.99 | 20.63 | 16.42 | 4.21 | 48.37 | 1148 |
| Pericardial cavity opened to atmospheric pressure. Rt. auricular pressure rose 3.5 cm. water. | 46.06 | 48.17 | 22.54 | 16.87 | 5.67 | 47.37 | 835 |
| Suction equivalent to 8 cm. water applied to pericardial cavity. Rt. auricular pressure fell 1 cm. below level of control. | 46.02 | 48.51 | 21.79 | 16.93 | 4.86 | 45.89 | 944 |
| | 48.01 | 49.50 | 22.10 | 17.19 | 4.91 | 44.91 | 916 |
| Positive pressure equivalent to 8 cm. water applied to pericardial cavity. Rt. auricular pressure rose 11 cm. above level of control. | 41.34 | 49.35 | 24.77 | 10.29 | 14.48 | 39.49 | 272 |
| | 39.92 | 47.41 | 25.41 | 10.02 | 15.39 | 43.32 | 281 |

TABLE I—CONT'D

**Experiment 5. Greyhound, weight 21 kg. Barbital sodium 6:3 gm. intravenously.**

| | Carbon dioxide content, per cent by volume, 1 c.c. of blood | | Oxygen content, per cent by volume, 1 c.c. of blood | | Arterial-venous difference | Oxygen consumption, c.c. per min. | Cardiac output, c.c. per min. |
|---|---|---|---|---|---|---|---|
| | Arterial | Venous | Arterial | Venous | | | |
| Control | 50.75 | 52.71 | 21.04 | 17.42 | 3.62 | 122.71 | 3389 |
| | 50.07 | 51.47 | 21.13 | 17.50 | 3.63 | 124.03 | 3416 |
| Pericardium opened to atmospheric pressure. Rt. auricular pressure rose 4.2 cm. | 50.01 | 52.08 | 23.49 | 17.73 | 5.76 | 113.83 | 1976 |
| Suction equivalent to 8 cm. water applied to pericardial cavity. Rt. auricular pressure fell to about 0.5 or 1 cm. above level of control. | 53.22 | 55.82 | 22.02 | 16.74 | 5.28 | 113.29 | 2145 |
| | 54.25 | 55.92 | 22.79 | 17.75 | 5.04 | 126.66 | 2513 |
| Positive pressure equivalent to 8 cm. water applied to pericardial cavity. Rt. auricular pressure rose to about 9 cm. water above level of control. | 49.55 | 53.37 | 23.34 | 16.81 | 6.53 | 123.96 | 1898 |
| | 49.63 | 52.64 | 23.63 | 16.72 | 6.91 | 111.55 | 1614 |

*Experiment 7.*—A police dog, weighing 21 kg. Two control determinations were done, but one giving a minute volume output of over four liters was discarded. Arterial blood: $CO_2$ – 47.38, $O_2$ – 21.36; venous blood: $CO_2$ – 47.54, $O_2$ – 17.83. Oxygen consumption per minute 105.20 c.c. Cardiac output per minute 2,980 c.c. The pericardial cavity was then opened to atmospheric pressure. Arterial blood: $CO_2$ – 46.95, $O_2$ – 21.33; venous blood: $CO_2$ – 48.73, $O_2$ – 17.34. Oxygen consumption per minute 101.03 c.c. Cardiac output per minute 2,532 c.c. Another determination was made. Arterial blood: $CO_2$ – 45.26, $O_2$ – 20.97; venous blood: $CO_2$ – 49.64, $O_2$ – 16.98. Oxygen consumption per minute 105.73. Cardiac output per minute 2,649 c.c. Suction equivalent to 9 cm. of water was applied to the pericardial cavity and minute volume output determinations of 2,613 c.c., 2,443 c.c., and 2,320 c.c. were obtained. Pressure equivalent to 9 cm. of water was applied to the pericardial cavity and a minute volume output determination of 1,301 c.c. was obtained. The temperature of this dog was held at 37° to 37.2° C. The minute volume output did not rise during the period in which suction was applied to the pericardial cavity. The explanation of this cannot be given.

*Experiment 8.*—A collie, weighing about 14 kg. Roentgenograms of the chest were taken. Arterial blood: $CO_2$ – 48.68, $O_2$ – 21.29; venous blood: $CO_2$ – 48.34, $O_2$ – 19.46. Oxygen consumption per minute 86.32 c.c. Cardiac output per minute 4,716 c.c. This seems to be too high for a dog of this weight. The pericardial cavity was opened to the pressure of the atmosphere. The right auricular pressure rose 4 cm. Arterial blood: $CO_2$ – 48.66, $O_2$ – 21.62; venous blood: $CO_2$ – 49.80, $O_2$ – 18.62. Oxygen consumption per minute was 80.28 c.c. Cardiac output per minute 2,676 c.c. Suction equivalent to 7 cm. of water was applied to the pericardial

cavity. The right auricular pressure fell to the level of the control. Arterial blood: $CO_2$ – 50.37, $O_2$ – 21.15; venous blood: $CO_2$ – 50.73, $O_2$ – 17.81. The oxygen consumption per minute was 88.01 c.c. The cardiac output per minute was 2,635 c.c. The suction was discontinued and atmospheric pressure was applied. Arterial blood: $CO_2$ – 49.78, $O_2$ – 20.18; venous blood: $CO_2$ – 51.22, $O_2$ – 15.28. The oxygen consumption per minute was 82.33 c.c. The minute volume output was 1,680 c.c. Positive pressure equivalent to 6 cm. of water was applied to the pericardial cavity.

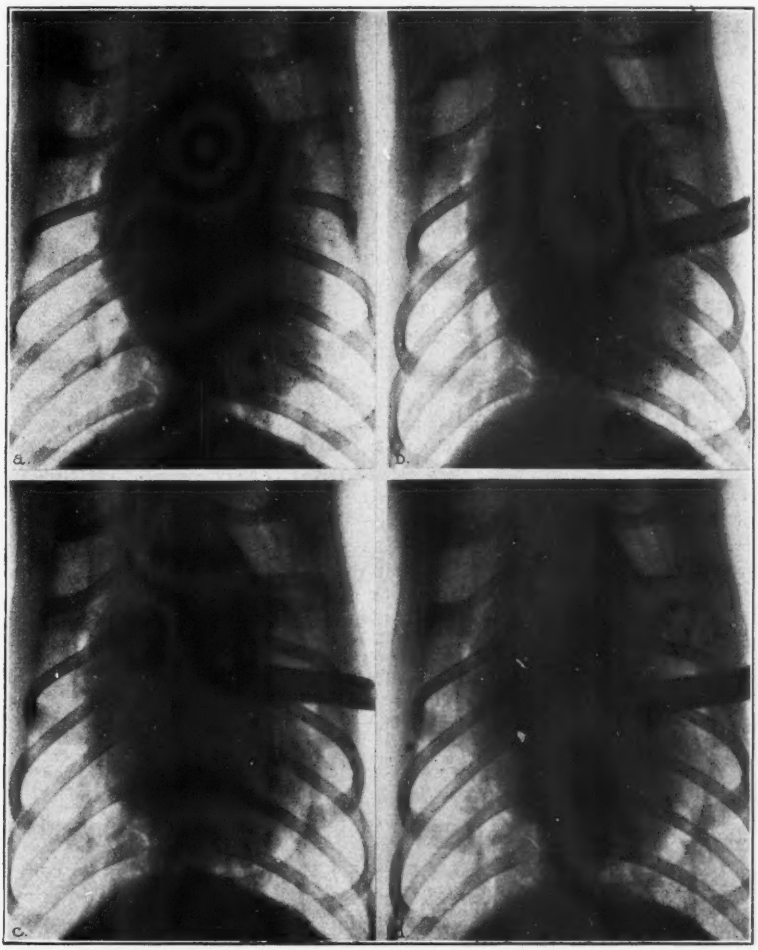



Fig. 3.—Roentgenograms showing effects of gas pressures in the pericardial cavity. *a* was taken before the pericardial cavity was opened to atmospheric pressure. It is similar to *b* taken later with a catheter in the pericardial cavity and a suction pressure equivalent to 7 cm. of water. *c* was taken after release of the suction, the intrapericardial pressure being equal to that of the atmosphere. The pericardium can be seen ballooned out away from the heart. *d* was taken with the intrapericardial pressure equivalent to that of atmosphere plus 6 cm. of water. The pericardium is ballooned out and the heart is compressed. The heart is definitely smaller than normal.

The right auricular pressure rose 6.5 cm. above the level of the control. Arterial blood: $CO_2$ – 48.54, $O_2$ – 20.78; venous blood: $CO_2$ – 53.03, $O_2$ – 13.36. The oxygen consumption per minute was 45.02 c.c. The cardiac output per minute was 607 c.c. Roentgenograms were taken of the chest with the end of the catheter inserted into the pericardial cavity. These roentgenograms were taken with the relative distances of target, dog and plate identical so that they might be compared one with another (Fig. 3).

*Experiment 9.*—A collie, weighing 13 kg. The determinations for the control and for the effect of atmospheric pressure are not given because they are not accurate. The following determinations, however, seem to be acceptable. The pericardial cavity was opened to atmospheric pressure and suction equivalent to 8 cm. of water was applied to the pericardial cavity through a catheter. Arterial blood: $CO_2$ – 45.34, $O_2$ – 21.87; venous blood: $CO_2$ – 46.71, $O_2$ – 16.48. The oxygen consumption per minute was 74.29 c.c. The cardiac output per minute was 1,378 c.c. Pressure equivalent to 8 cm. of water was applied to the pericardial cavity through a catheter. Arterial blood: $CO_2$ – 39.28, $O_2$ – 23.27; venous blood: $CO_2$ – 45.94, $O_2$ – 14.56. The oxygen consumption per minute was 55.48 c.c. The cardiac output per minute was 639 c.c. Negative pressure equivalent to 8 cm. of water was again applied to the pericardial cavity. Arterial blood: $CO_2$ – 43.11, $O_2$ – 21.10; venous blood: $CO_2$ – 47.31, $O_2$ – 13.62. The oxygen consumption per minute was 78.65 c.c. The cardiac output per minute was 1,051 c.c.

*Experiment 10.*—A mongrel, weighing 19 kg. The control determination was as follows: Arterial blood: $CO_2$ – 44.70, $O_2$ – 14.97; venous blood: $CO_2$ – 45.01, $O_2$ – 12.13. The oxygen consumption per minute was 69.53 c.c. The cardiac output per minute was 2,448 c.c. The pericardial cavity was opened to atmosphere. The right auricular pressure rose 3 cm. of water. Arterial blood: $CO_2$ – 43.48, $O_2$ – 14.96; venous blood: $CO_2$ – 45.09, $O_2$ – 11.16. The oxygen consumption per minute was 64.48 c.c. The cardiac output per minute was 1,696 c.c. A catheter was sutured into the pericardial cavity and suction equivalent to 10 cm. of water was applied. The right auricular pressure rose to the level of the control. Arterial blood: $CO_2$ – 43.53, $O_2$ – 14.29; venous blood: $CO_2$ – 45.19, $O_2$ – 11.26. The oxygen consumption per minute was 64.19. The cardiac output per minute was 2,111 c.c. Positive pressure equivalent to 8 cm. of water was applied to the pericardial cavity. The right auricular pressure rose to 8 cm. above the level of the control. Arterial blood: $CO_2$ – 42.53, $O_2$ – 15.28; venous blood: $CO_2$ – 44.71, $O_2$ – 11.48. The oxygen consumption per minute was 54.40 c.c. The cardiac output per minute was 1,431 c.c.

*Experiment 11.*—A police dog, weighing 18.2 kg. The control determination was as follows: Arterial blood: $CO_2$ – 53.15, $O_2$ – 14.40; venous blood: $CO_2$ – 54.46, $O_2$ – 11.47. Oxygen consumption per minute 82.46 c.c. Cardiac output per minute 2,814 c.c. The pericardial cavity was opened to the atmosphere. Arterial blood: $CO_2$ – 50.34, $O_2$ – 14.67; venous blood: $CO_2$ – 50.21, $O_2$ – 9.66. Oxygen consumption per minute 86.09 c.c. Cardiac output per minute 1,718 c.c. A catheter was placed in the pericardial cavity and negative pressure equivalent to 10 cm. of water was applied. The arterial blood: $CO_2$ – 49.69, $O_2$ – 14.13; venous blood: $CO_2$ – 52.77, $O_2$ – 10.03. The oxygen consumption per minute was 87.99 c.c. The cardiac output per minute was 2,146 c.c.

*Experiment 12.*—Control determinations were as follows: Arterial blood: $CO_2$ – 45.70, $O_2$ – 14.40; venous blood: $CO_2$ – 48.49, $O_2$ – 11.07. The oxygen consumption per minute was 71.22 c.c. The cardiac output per minute was 2,139 c.c. Another determination was made. Arterial blood: $CO_2$ – 44.65, $O_2$ – 14.85; venous

blood: $CO_2$ – 47.39, $O_2$ – 12.24. The oxygen consumption per minute was 61.82 c.c. The cardiac output per minute was 2,368 c.c. The pericardial cavity was then opened to the atmosphere; respiration became more rapid and more shallow. The arterial blood: $CO_2$ – 44.78, $O_2$ – 15.37; venous blood: $CO_2$ – 47.66, $O_2$ – 10.35. The oxygen consumption per minute was 61.89 c.c. The cardiac output per minute was 1,233 c.c. The dog was then placed in the pressure chamber. When negative pressure was applied, respiration became slower and deeper as compared to respiration when the intrapericardial pressure was that of atmosphere. When the pressure in the chamber was increased to a level above that of the atmosphere, respiration became shallow and rapid. A pressure equivalent to 15 cm. of water above that of atmosphere killed the dog. The specimens of venous blood clotted so that cardiac output determinations cannot be given.

*Experiment 13.*—A bulldog, weighing 16.4 kg. A control determination was carried out. Arterial blood: $CO_2$ – 58.12, $O_2$ – 18.25; venous blood: $CO_2$ – 58.99, $O_2$ – 15.83. The oxygen consumption per minute was 75.07 c.c. The cardiac output per minute was 3,102 c.c. The pericardial cavity was opened to the atmosphere. The right auricular pressure rose 3 cm. of water above the level of the control. Arterial blood: $CO_2$ – 51.64, $O_2$ – 19.05; venous blood: $CO_2$ – 54.05, $O_2$ – 14.93. The oxygen consumption per minute was 76.66. The cardiac output per minute was 1,860 c.c. A catheter was sutured into the pericardial cavity and suction of 6 cm. of water was applied. The right auricular pressure fell to the control level. Arterial blood: $CO_2$ – 49.65, $O_2$ – 19.96; venous blood: $CO_2$ – 51.72, $O_2$ – 15.17. The oxygen consumption per minute was 91.02 c.c. The cardiac output per minute was 1,900 c.c. Positive pressure equivalent to 6 cm. of water was applied to the pericardial cavity. The right auricular pressure rose to about 6 cm. of water. After this pressure had been applied for about one hour the following determination was obtained. Arterial blood: $CO_2$ – 42.09, $O_2$ – 22.42; venous blood: $CO_2$ – 51.25, $O_2$ – 7.99. The oxygen consumption per minute was 43.33 c.c. The cardiac output per minute was 300 c.c. Suction equivalent to 6 cm. of water was applied to the pericardial cavity and about 30 minutes later the following determination was obtained. Arterial blood: $CO_2$ – 46.70, $O_2$ – 19.82; venous blood: $CO_2$ – 50.43, $O_2$ – 12.14. The oxygen consumption per minute was 105.08 c.c. The cardiac output per minute was 1,368 c.c.

TABLE II*

| | Carbon dioxide content, per cent by volume, 1 c.c. of blood | | Oxygen content, per cent by volume, 1 c.c. of blood | | Arterial-venous difference | Oxygen consumption, c.c. per min. | Cardiac output, c.c. per min. |
|---|---|---|---|---|---|---|---|
| | Arterial | Venous | Arterial | Venous | | | |
| CONTROL | 50.96 | 52.74 | 21.74 | 18.51 | 3.23 | 86.33 | 2871 |
| Pericardium open to atmospheric pressure | 47.42 | 49.85 | 23.30 | 18.25 | 5.05 | 81.60 | 1831 |
| Negative pressure applied to pericardial cavity | 49.17 | 51.42 | 22.05 | 17.70 | 4.35 | 81.87 | 2084 |
| Positive pressure applied to pericardial cavity | 44.30 | 49.45 | 24.94 | 15.25 | 9.69 | 77.49 | 1026 |

*Average measurements for cardiac output studies of the five experiments recorded in Table I. In these experiments the temperature of the dog was kept constant and this eliminated certain fluctuations in the measurements.

*Discussion.*—It is apparent from the above experiments that the minute volume output of the heart decreases when the pressure on the heart and the intrapericardial portions of the great vessels at the base of the heart is changed from the usual negative pressure of the thorax to the pressure of the atmosphere. The five experiments recorded above in tabular form give the following average measurements (Table II) for the minute volume output: (1) Control 2,871 c.c.; (2) the pericardial cavity opened to atmosphere, 1,831 c.c., a decrease of 36 per cent; (3) negative pressure applied to the pericardial cavity, 2,084 c.c., a restoration of only 25 per cent of the decrease; (4) positive pressure applied to the pericardial cavity, 1,026 c.c., a decrease of 64 per cent from the level of the control. These values are shown graphically in Fig. 2. In recovery experiments Beck and Cox found that without anesthesia the decrease in minute volume output measured from 15 to 30 per cent when the pericardial cavity was opened to atmospheric pressure. The effect of pneumocardiac tamponade is definitely greater in the dog anesthetized with sodium barbital. The average decrease in the experiments with this drug was 36 per cent. This may indicate that the effect of pneumocardiac tamponade was proportionately greater when the circulation had deviated from the normal as presumably occurred under anesthesia with this drug. Such reasoning is in agreement with clinical experience. In the human being, the normal heart satisfactorily withstands exposure at operation. If the heart has only a relatively slight reserve power, however, the effect of pneumocardiac tamponade is relatively greater because under such circumstances the cardiac action may fail completely when the heart is exposed to atmosphere. Negative pressure applied to the pericardial cavity after the latter had been opened to the exterior brought about an increase in the minute volume output of the heart. The average measurements of this restoration of output was 25 per cent. In only one experiment was the normal control level approximately restored by negative pressure.

The effect of positive intrapericardial pressure varied with the degree of pressure and the duration of its application. A fall of 63 per cent from the control level was obtained by the application of pressure equivalent to 7, 8, or 9 cm. of water applied during a total period of thirty to forty-five minutes. Another point for consideration brought out by the experiments was the slow rate of recovery in cardiac output after positive pressure had been applied to the intrapericardial structures. For that reason the determination of the effect of positive pressure was reserved usually as the final step of the experiment. A study of the rate of circulatory recovery, after tamponade has been established, merits further work because it carries a practical application. It will afford an explanation, we believe, for those fatalities in thoracic surgery that occur hours or even a day or two after the

thoracic wound has been closed. In other words, the effect of pneumocardiac tamponade may persist for hours after the tamponade has been corrected, and if the heart cannot make the added effort to overcome this burden it may after a long struggle completely fail.

It is seen from the average values in the above table that the carbon dioxide content of both arterial and venous blood was highest for the normal control and was progressively decreased, in the following order, by the application of negative pressure, atmospheric pressure and finally positive pressure in the pericardial cavity. The oxygen content of arterial blood was lowest in the control. It was slightly higher when negative pressure was applied to the pericardial cavity and it progressively increased with atmospheric pressure and positive pressure. The oxygen content of venous blood was highest in the normal control and was progressively decreased by atmospheric pressure, negative pressure and positive pressure applied to the pericardial cavity. The difference in oxygen content of arterial and venous blood was lowest in the normal control and was progressively increased by negative pressure, atmospheric pressure and positive pressure in the pericardial cavity. The oxygen consumption per minute was highest in the normal control and became progressively less with negative pressure, atmospheric pressure and positive pressure in the pericardial cavity.

There occurred almost uniformly an increase in the respiratory rate and a slight decrease in the respiratory excursion when the pericardial cavity was opened to atmosphere. These changes were corrected partially by the application of negative pressure in the pericardial cavity and were greatly exaggerated when positive pressure was applied to the pericardial cavity.

It was also noted that the pressure in the right auricle increased when the pericardial cavity was opened to atmosphere. This increase in pressure measured 3 to 4 cm. of water. It was corrected by the application of negative pressure equivalent to 6 or 8 cm. of water. The intraauricular pressure also rose when positive pressure was applied to the pericardial cavity.

### PRESSURE DETERMINATIONS

A series of experiments was carried out to show the effects upon arterial, venous and intrapleural pressures brought about by the application of different gas pressures in the pericardial cavity. The following experiments illustrate the changes that occurred.

*Experiment 14.*—A mongrel dog, weighing 18.9 kg., was anesthetized with ether Jan. 14, 1931, and the pericardium was sutured to the left thoracic wall. On Feb. 4, 1931, this dog was anesthetized with barbital sodium, 5.5 gm., given intravenously. The left carotid artery was connected to a mercury manometer; the superior vena cava was connected to a Marey tambour through the jugular vein,

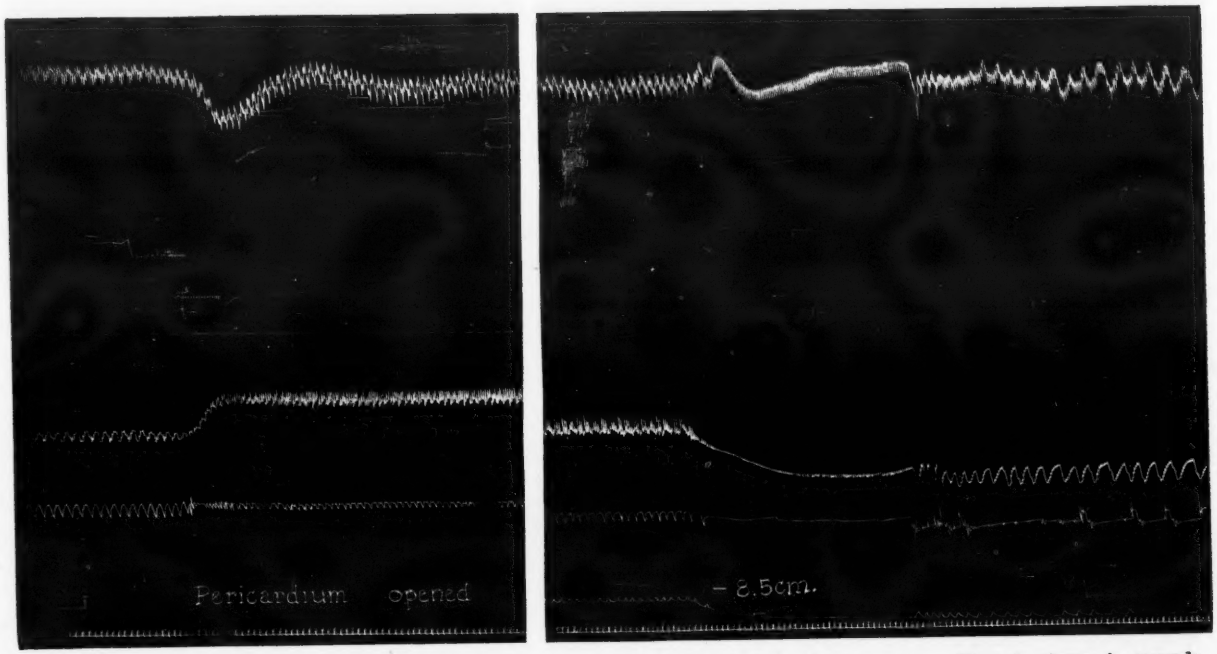



**Fig. 4.—The tracings from above down represent arterial, venous, intrapleural and chamber pressures. Time is shown in seconds.**

and the pleural cavity was connected to an air tambour. Graphs of arterial, venous and intrapleural pressures were obtained. An incision was then made over the scar of the operation and the pericardial cavity was exposed to the pressure of the atmosphere.

When the pericardial cavity was opened to atmosphere there occurred (Fig. 4): (1) An immediate fall in arterial pressure from 116 to 96 mm. of mercury. The arterial pressure recovered its former level in 15 seconds and continued at the level of 108 mm. of mercury. Coincidentally with the fall in blood pressure there occurred a fall in pulse rate from 183 to 162 per minute. The pulse rate gradually came back to the former level. (2) A sustained rise in venous pressure and an accentuation of the venous pulsation. The rise in venous pressure was equivalent to the pressure of 3 or 4 cm. of water. (3) An increase in the respiratory rate from 42 to 51 per minute and a decrease in the amplitude of respiratory excursion. The intrapleural pressure curve showed little if any alteration in level. As measured by a water manometer the respiratory excursion before opening the pericardial cavity varied from −8.12 to −1.8 with a mean pressure of −4.9 cm. After the pericardial cavity was opened to atmosphere the fluctuation in pressure varied from −6.4 to −1.8 with a mean pressure of −4.1 cm. The measurement of −1.8 represented the intrapleural pressure at the position of rest; namely, at the end of expiration, and this pressure was not altered by opening the pericardium.

The dog was then inserted into the negative chamber and a pressure equivalent to 8.5 cm. of water below that of atmosphere was applied. When this pressure was applied the following observations were made (Fig. 4): (1) The arterial pressure rose from about 110 to 120 mm. of mercury, then there was a slight decrease and then a pressure maintained at the level of about 116 mm. of mercury. Respiratory waves in the arterial pressure were absent for a period of 37 seconds. The pulse rate did not change. (2) The venous pressure fell to a level slightly below that of the control. Respiratory waves were absent temporarily from the venous pressure tracing. The amplitude of the venous pulsation decreased. (3) A period of apnea was present for 37 seconds. Respiration then became somewhat irregular and slightly deeper. The respiratory rate decreased from 42 to 24 per minute. The respiratory excursion measured −12 to −5 cm. of water, with a mean pressure of −8.5 cm. This represented an increase in the intrapleural pressure. The pressure in the chamber was less; i.e., more negative, than was the intrapleural pressure of the control. Both the venous and the intrapleural pressures fell below the control levels when negative pressure was applied to the chamber. The negative pressure was released and atmospheric pressure was applied. The arterial pressure showed no change in level and the pulse rate remained about the same. The venous pressure rose and remained elevated. Respiration then became more regular, more rapid and less deep. The intrapleural pressure fluctuated from −7.4 to 0 cm. of water, giving a mean pressure of −3.7 cm.; i.e., somewhat higher or less negative than when negative pressure was applied.

The pressure in the chamber was then increased to the equivalent of 8 cm. of water and later to 14 cm. of water (Fig. 5). The general level of the arterial pressure did not fall and the pulse rate did not change. The venous pressure rose markedly and the pulsation increased in amplitude. The respiratory rate had increased to about 100 per minute. The amplitude of respiration measured from 0 to −4 cm. of water. The intrapleural pressure was decreased.

Pressure determinations taken from another experiment are shown in Fig. 6. When the pericardial cavity was opened to atmospheric pressure, there occurred a fall of 24 mm. of mercury in arterial pres-

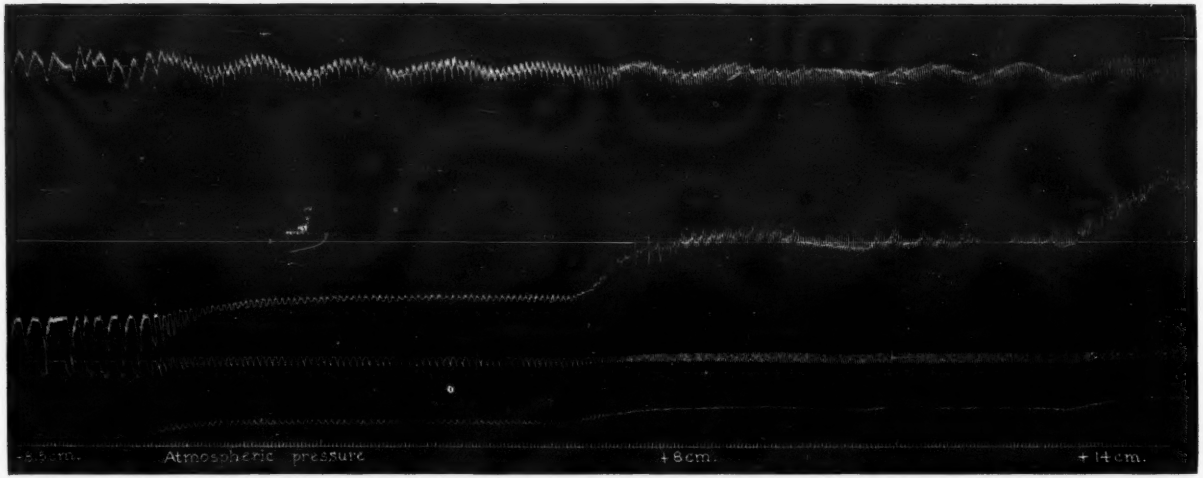



Fig. 5.—The tracings from above down represent arterial, venous, intrapleural and chamber pressures. Time is shown in seconds. Although the minute volume output of the heart was reduced by the application of positive pressure to the intrapericardial structures the general level of arterial pressure was maintained. The stimulus to the vasomotor center was by relative anoxemia as indicated by the dearth of oxygen in the blood returning to the heart.

sure. The arterial pressure rose in a few seconds but it did not quite recover its former level. The pulse rate showed a slight rise. The venous pressure became elevated and the pulsation increased in amplitude. The respiratory rate increased from 34 to 39 per minute, the amplitude decreased slightly and the intrapleural pressure became slightly less negative.

The dog was inserted into the chamber and the pressure was reduced to a point equivalent to 10 cm. of water. Inasmuch as the pericardial cavity communicated with the exterior, this negative pressure was applied to the intrapericardial structures. There occurred a slight transient rise in the arterial pressure and an increase in the pulse rate. The venous pressure fell and the amplitude of the pulsation decreased. The respiratory rate became slower. The period of apnea shown in the preceding experiment did not occur. The pressure in the chamber was then changed to that of the atmosphere and later an increment of pressure equivalent to 10 cm. of water was applied. With the application of positive pressure there developed accentuated pulsations of arterial, venous and intrathoracic pressures. The arterial pressure showed little change but had the positive pressure been continued over a longer period of time it would have fallen. With the application of 10 cm. of pressure the pulse rate decreased. The venous pressure showed a marked rise.

*Discussion.*—When the pericardial cavity was opened to the pressure of atmosphere there occurred a slight but usually transient fall in the arterial pressure. Occasionally the arterial pressure did not recover fully its former level. The average fall in arterial pressure was about 20 mm. of mercury. The pulse rate usually showed a slight increase. The venous pressure increased and the amplitude of the venous pulse also increased. These changes in venous pressure and pulse were sustained. At the same time the respiratory rate usually increased and the depth of respiratory movement decreased. The intrapleural pressure usually, but not always, became slightly less negative. This slight elevation in intrapleural pressure possibly could be accounted for by a decrease in the depth of respiration.

When the dog was placed in a negative chamber and the pressure was reduced by about 10 cm. of water the arterial pressure occasionally showed a slight rise but the general level of pressure showed little if any change. The venous pressure returned to the normal level as did also the amplitude of pulsation. Usually a period of apnea appeared when negative pressure was applied. Respirations became slower and deeper and not infrequently were coupled in groups of two or three. The intrapleural pressure was made more negative by a pressure of –10 cm. of water in the chamber.

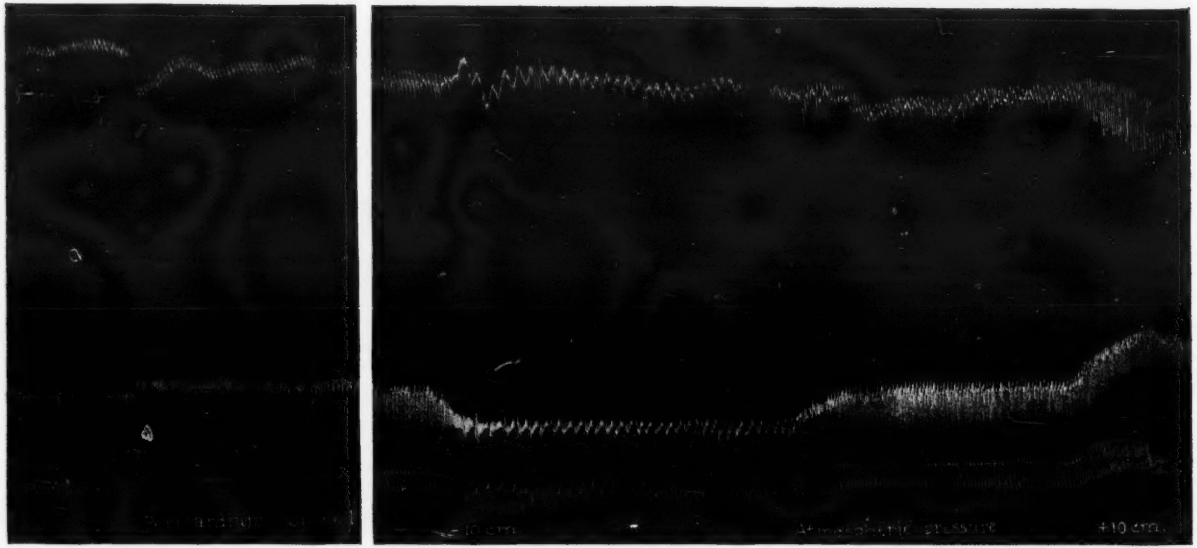


Fig. 6.—The tracings from above down represent arterial, venous and intrapleural pressures. The alteration in the circulation brought about by opening of the pericardial cavity to atmosphere is qualitatively the same as the compression effect exerted by positive intrapericardial pressure.

When positive pressure equivalent to 10 cm. of water was applied to the chamber and to the intrapericardial structures, the arterial pressure fell only after the pressure had been applied several minutes. This maintenance of arterial pressure was a striking example of the effect of vasomotor control. The venous pressure rose and the pulsations became greatly exaggerated. Respiration became deep and rapid. With smaller increments of positive pressure the intrapleural pressure usually became less negative.

We may attempt to give an explanation for the above changes. The transient but immediate fall in arterial pressure occurring when the

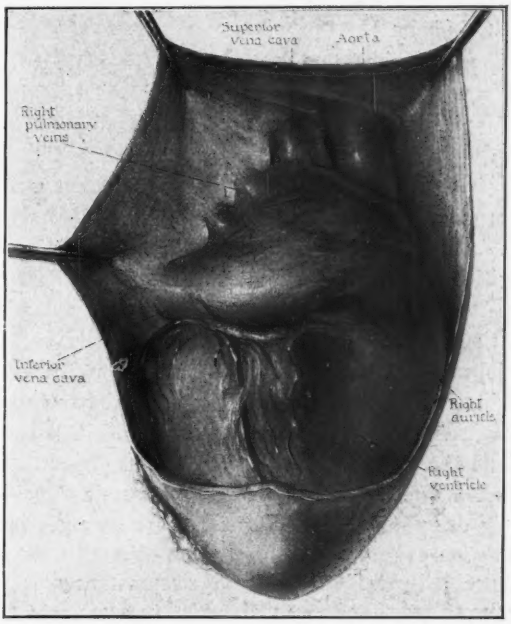



Fig. 7.—The intrapericardial segments of the great vessels at the base of the heart. When the pericardial cavity is opened to the atmosphere the impairment to the circulation is brought about by the pressure effect exerted primarily upon the venae cavae and the right auricle.

pericardial cavity was open to atmosphere was due to an impairment in the filling of the heart. It required about half a minute for blood to accumulate in the venous system and to assimilate sufficient pressure to "break through" this partial obstruction presented by the pressure of the atmosphere upon the venae cavae (Fig. 7). The minute volume output of the heart having been reduced 36 per cent, one would expect the arterial pressure to have shown greater changes. The arterial pressure was maintained even after the cardiac output

was reduced still further by the application of positive pressure. The analyses show no increase in the carbon dioxide content of the blood; indeed, the carbon dioxide content was actually decreased. This decrease in carbon dioxide content of the blood was brought about by the increase in respiratory rate occurring when the pericardial cavity was opened to atmosphere or when positive pressure was applied to the intrapericardial structures. Under these circumstances the oxygen content of the blood returning to the heart was abnormally low and this decrease in oxygen content presumably furnished the stimulus to both respiratory and vasomotor centers.

No better explanation of the venous pressure changes can be given than that of Cohnheim:[3]

"Yet, although the resistance offered by the pericardium is overcome by the venous pressure, the amount of blood reaching the heart from the veins in the unit of time is of necessity diminished, and that in proportion to the resistance, i.e., to the tension in the pericardium. Under these circumstances the venous flow suffers not merely a quantitative but a *qualitative* change; *the venous pulse becomes much more evident than is normally the case.* For although it is perfectly true that, even with a normal circulation, the veins which are situated in proximity to the heart display pulsations, synchronous with, and depend upon, the auricular systole, these are usually so feeble as to require special methods for their demonstration; and since the auricle does not during its systole, still less during its diastole, offer any resistance worth mentioning to the instreaming blood, it is no essential departure from the truth to say that the venous stream, despite these pulsations, is uniform and continuous. The case is altered of course, when the flow of venous blood into the heart becomes impeded; for every aggravation or weakening of the impediment must make itself much more decidedly felt under such circumstances. If, now, the pericardium be so overfilled as to give rise to somewhat considerable tension, the venous blood may cease to enter at the moment when the abnormal resistance is augmented by the normal auricular contraction; and may enter only during the auricular diastole. The venous stream is then decidedly rhythmical, and not only may this character be observed with the greatest ease in the column of the soda-manometer [soda manometer is for venous pressure], but there is also perceptible a *rhythmical turgescence* of the visible veins, most striking, of course, in proximity to the heart."

In experiments that we carried out, the pressure in the pericardial cavity was about the same as the intrapleural pressure. Occasionally the intrapericardial pressure measured 0.5 cm. of water less than the intrapleural pressure. Both of these pressures originate from the

elastic recoil of the lungs, and this similarity of pressures was to be expected.

The changes in the intrapleural pressure recorded in the experiments were produced chiefly by the enlargement of, or by the compression of, the thoracic cavity brought about by the gas pressure in the chamber. Such changes in the size of the thorax were easily demonstrated on the dead animal. To avoid this complication and its possible effect upon the blood flow through the lungs the cardiac output determinations were carried out without using the chamber, the intrapericardial pressure being altered by means of a catheter hermetically sutured in the wound. Even with this method the pericardium, ballooned out with air (Fig. 3), may encroach slightly upon the lungs, but we believe that this in itself was of little or no significance in changing the pulmonary circulation.

The mechanism that brings about the circulatory changes of pneumocardiac tamponade is a pressure phenomenon. The gas pressure acts in the same manner as does effusion of fluid in the pericardial cavity. The various manifestations of pneumocardiac tamponade are relieved by the application of negative pressure to the pericardial cavity. In our experiments the degree of improvement brought about by the application of negative pressure was usually incomplete although in one experiment the cardiac output was brought back to the normal level.

## CLINICAL APPLICATION

The clinical application of the measurements presented here is apparent to the surgeon who does thoracic work. That pneumocardiac tamponade may be a factor in bringing about circulatory failure there is no doubt. The patient with a good circulation can withstand the effect of atmospheric pressure upon the heart. If the circulatory reserve is restricted, however, pneumocardiac tamponade cannot be tolerated.

The question of the negative chamber was settled some years ago by surgeons interested in thoracic surgery. The few negative chambers constructed in the early part of this century have been dismantled. In so far as we are aware there is no surgeon today who does thoracic surgery in the negative chamber.

In view of our measurements, we believe that this question should be reconsidered. Indeed, from our present point of view we would like to advocate its use in thoracic surgery.

## REFERENCES

1. Beck, C. S., and Cox, W. V.: The Effect of Pericardiostomy on the Mechanics of the Circulation, Arch. Surg. 21: 1023-1039, 1930.

2. Sauerbruch, F.: Ueber die physiologischen und physikalischen Grundlagen bei intrathoracalen Eingriffen in meiner pneumatischen Kammer, Verhandl. d. deutsch. Ges. f. Chir. 33: 1904.
3. Cohnheim, Julius: Lectures on General Pathology; a Handbook for Practitioners and Students. Section I, The Pathology of the Circulation. Translated by A. B. McKee, London, New Sydenham Society, 1889, Vol. 1, p. 26.

## DISCUSSION

DR. D. C. ELKIN.—Dr. Beck's excellent work on this subject brings up a very important clinical fact which he mentioned at the end, reciting the case of the patient who died, and I would like to put into the record a similar experience, where an operation was done in order to give more room in the chest for a large rheumatic heart. A portion of the third, fourth and fifth ribs and the costal cartilages were removed under procaine anesthesia. The pleura was not opened. The operation was done without any difficulty. The patient's arterial pressure dropped constantly for thirty-six hours and the patient died.

There was nothing in the operation to account for his death. The autopsy showed nothing to account for it and I feel that possibly in these patients, with a low cardiac reserve—that the added "bump" as Dr. Beck calls it, to the circulation of the atmospheric pressure on the great vessels at the base of the heart may be the cause of death. Unfortunately, in our case, we were not thinking of this. We did not make any readings of venous pressures, which should always be done, I think, in order to see whether the venous pressure is rising with the drop in arterial pressure. It may be, as Dr. Beck suggests, that the Sauerbruch negative chamber has been abandoned too quickly, and has a very definite place in cardiac surgery.

DR. LILJENCRANTZ (OAKLAND, CALIFORNIA).—Professor Sauerbruch's negative pressure chamber was abandoned in his own clinic, I believe, because of its complexity and awkwardness; and because in the positive-pressure gas-oxygen-ether apparatus as now used, a far simpler and more practical means of maintaining the intrapleural-atmospheric pressure differential is at hand. In commenting on this in his "Chirurgie der Brustorgane," Sauerbruch remarks that whereas, in the great majority of cases in which the pleural cavity is opened, positive pressure anesthesia proves satisfactory, there remains that in a small group of patients with narrow cardiac reserve, the original negative pressure chamber provides by far the wider margin of safety, and fatalities have resulted from failure to utilize it.

Sauerbruch offers no explanation of this phenomenon, and it is to this question that Dr. Beck has just given us the most reasonable answer. Whether positive or negative pressure methods of anesthesia are used should make little difference in the satisfactory maintenance of the atmospheric-intrapleural differential. It is obvious, however, that whereas with negative pressure anesthesia the atmospheric-*intrapericardial* differential would be comparatively little disturbed, no provision whatsoever for its maintenance is afforded by a system in which the thorax is opened to atmospheric pressure, and respiration is maintained by increased intrabronchial pressures. It is exactly this disregard of intrapericardial pressure relationships that Dr. Beck has shown to be of such great consequence.

DR. REICHERT.—Dr. Beck mentioned that his results in cardiac output were not satisfactory until the last six weeks. Is that correct?

DR. BECK.—Yes.

DR. REICHERT.—And that he found the room temperature was a very important factor. I think our experimental work on the sympathetics has gone along sufficiently now to make us realize that we must consider room temperature and

the state of the animal as well as the patient in evaluating the effect of the sympathetic system on the organism.

For instance, if a patient or animal with a sympathectomized limb is kept in a room of 24° C., or more, you will usually find no difference in skin temperature. But if you put that animal or patient in a cool room, where it is 13° or 14° C. you can practically get any degree of difference ranging from ten to fifteen degrees of centigrade, between these two limbs. We did not find that out until recently and it has been applied clinically by Morton and Scott in the Evaluation of Sympathetic Nervous System and Upon Organisms in certain diseases. So our experiment will work continually with blood cardiac output and all these determinations. I think it is very important that we have all of our controls and observations in the same room temperature.

DR. BECK.—Discussions of pneumothorax usually concern the aeration of the lung. I should like to emphasize the point that the effect of pneumothorax upon the lungs is quite distinct from its effect upon the heart and the great vessels at the base of the heart. Although the lungs can be distended by positive pressure applied through the trachea, the effect of atmospheric pressure upon the venae cavae and the heart still persists. Indeed, it seems that if the lung is forcibly distended by pressure from without there may be an added increment of pressure upon these structures exerted through the distended lung, over and above the pressure of the atmosphere. Positive pressure insufflation of the lung, therefore, does not take care of this mechanism.

In our experiments the pericardial cavity only was opened to the atmosphere. By this method the effect of atmospheric pressure upon the circulation could be separated from its effect upon the lung. It is scarcely necessary to state that the circulatory changes, such as we have studied and measured, are a part of the physiological changes that occur when the pleural cavity or the mediastinum is opened without opening the pericardial sac. Indeed, I believe that in certain cases the circulatory changes brought about when the chest is opened are due more to pneumocardiac tamponade than to the collapse of lung. The circulatory failure or the shock that occasionally accompanies or follows an operation on the thorax, has been the subject of considerable study and discussion. The various factors that have been ascribed to the production of thoracic shock are collapse of lung, displacement of the mediastinum with encroachment of the lung on the opposite side, displacement of the heart with kinking of the venae cavae, mediastinal flutter, pleural reflex, loss of blood, loss of heat and infection. Sometimes the patient goes into shock when none of the various factors seem to have been the cause of it. Experience has taught surgeons to fear the performance of a thoracic operation on a patient with a poor circulation. I believe I am quoting Dr. Evarts Graham correctly in saying that in his experience "many of the thoracic surgical deaths are circulatory; in fact most all of them are if we exclude the acute infections. Certainly practically all of the unexpected deaths that I have encountered have been due to pulmonary edema."

Not many years ago the negative chamber versus positive pressure insufflation was a subject that received considerable discussion. About ten or fifteen years ago the question apparently was settled. The few negative chambers that had been constructed were dismantled and the subject dropped out of the literature. Although his chamber went to the heap of old iron, Dr. Willy Meyer's spirit of championship was readily fanned into flame when he heard of our studies and measurements. A recent letter states: "It always was and still is my opinion also in other than heart diseases that the negative chamber might still have its place when the surgeon is obliged to operate for intrathoracic troubles on very weak patients. . . . I regretted this occurrence a great deal (i.e., the dismantling of the

chamber at the Lenox Hill Hospital), and so expressed myself, adding that I still believe that looking at it from the point of benefit to the patient the negative pressure chamber has still, and will forever, have a place in the chapter of intrathoracic surgery.''

I believe the accumulated experience in thoracic surgery obtained during the past ten or fifteen years with positive pressure insufflation, amply justifies a reconsideration of the negative chamber. Pneumocardiac tamponade is a factor, indeed in certain cases it is the most important factor, in the production of thoracic shock and experience beckons for its correction.

# DECOMPRESSION OF THE HEART*

## (Case Report)

William A. Hudson, M.D.
Detroit, Michigan

THE patient, Mr. S., whose case is herewith reported, is a white male, nineteen years of age. He is of Swedish extraction and by occupation a garage helper.

He was in good health until March, 1930, at which time he developed a cough which was productive of whitish phlegm. At the same time he had pain in his left shoulder and over the precordium. The diagnosis at that time was acute rheumatic fever. His treatment consisted largely of bed rest and digitalis. During the course of this illness evidence of pleural, pericardial, peritoneal and joint effusions was noted. During the summer he was up and about, but unable to work.

September 30, 1930, this patient was admitted to Grace Hospital, complaining of swelling of his legs, shortness of breath, and general weakness. Physical examination revealed marked swelling of the lower extremities with cyanosis of all the nail beds. The pulse was 140, respiration 36, and blood pressure 126/112. There was a slight systolic indrawing of the intercostal interspaces and of the costal margin on the left. The apex beat could not be seen or felt. There was dullness 4½ cm. to the right and 11 cm. to the left of the sternal margin. A loud friction rub was heard best over the lower end of the sternum lasting through systole. The liver was felt four fingerbreadths below the costal margin. Electrocardiographic studies revealed an inversion of the T-waves in all leads. The conduction time was normal. Rotation tests showed no shifting of the cardiac axis. X-ray studies showed a much enlarged heart shadow of the leather bottle type. The diagnosis at this time was polyserositis, chronic with pericardial adhesions. On October 9, the tonsils, being moderately septic, were removed under local anesthesia. During the next ten days no change in any findings were noted. It was then decided to remove the bony cage over the heart, beginning at the base of the heart and working toward the apex, hoping in that way to obtain a maximum of benefit from any relaxation of the chest wall. Accordingly, on October 20, under novocaine anesthesia, through an incision extending along the left margin of the sternum from the third rib down to the fifth, thence lateralward along the fifth to the midaxillary line, a skin and soft tissue flap was raised and the fourth rib exposed. This rib, with its costal cartilage and periosteum, was removed to the nipple line. During this operation it was noted that as soon as the underlying structures were released from the fourth rib that the cardiac impulse over the third and fourth interspaces was very much increased. The skin and soft tissue flap was then replaced.

On October 27 the pulse was 114, blood pressure 130/70. The patient was feeling better and he was sleeping better. He now had one new complaint, this was of a pounding and throbbing in his head. On this day, the skin and soft tissue flap was again raised and a section of the fifth rib, together with its costal cartilage and periosteum, was removed from the sternum to the anterior axillary fold. The same increase in the cardiac impulse was noted in the fifth interspace, that had

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Read at the Fourteenth Annual Meeting of the American Association for Thoracic Surgery at San Francisco, July 1, 2, 3, 1931.

previously been noted over the third and fourth interspace. The patient showed no new symptoms and was permitted to be up in a wheel chair. On November 8 the incision was again opened under novocaine anesthesia and sections of the sixth and seventh ribs, together with costal cartilage and periosteum, were removed to the anterior axillary line. It was noted that there was a precordial impulse extending over the entire area from which the bony support had been removed. A considerable amount of induration developed about the site of the operation, but as time went on this gradually softened and the patient became more and more active. He was discharged from the hospital January 31, 1931, with a pulse of 90, blood pressure 100/68. He has been seen since that time at frequent intervals. There has been noted some swelling of feet when he is on his feet any length of time. His x-ray studies reveal a decrease in the size of the heart shadow and his electrocardiographic studies show a return to normal of the T-waves, but rotation tests show no shifting of the cardiac axis. It would appear that the mediastinum is fixed.

We would call special attention to the fact that our operative procedures have dealt first, with the bony frame over the base of the heart and then finally with that over the apex; second, we are inclined to favor multiple stage operation rather than a single stage operation for the removal of this bony frame, hoping in that way to permit a gradual readjustment of the intramediastinal and intrapleural relationships; finally, we may later find it necessary to remove a portion of the sternum, or to open the pericardium and release adhesion, or to remove portions of the pericardium as has previously been reported before this Association.

It is our belief that, if any or all of the latter procedures become necessary, this patient will be a better subject for future operations because of the relief he has thus far obtained. Furthermore, the operative procedures, so far carried out, have been planned in such a manner that they pave the way for later operations, should one or more become necessary.

We wish to express to Doctor Norman Clark our appreciation of his close cooperation.

## DISCUSSION ON PAPERS BY DR. ELKIN AND DR. PHILLIPS AND DR. HUDSON

DR. E. F. BUTLER.—Although reports of operations upon the heart are increasing in frequency, cardiac surgery is still in its infancy. Three different problems have been presented by Dr. Beck, Dr. Elkin and Dr. Hudson. The problems involved in the surgical treatment of hearts damaged by long-continued disease are still obscure. It is to be hoped that these case reports will stimulate those engaged in experimental and clinical research to seek additional methods for accurately determining the operability of such cases.

DR. CLAUDE S. BECK.—I would like to congratulate Drs. Elkin and Phillips and Dr. Hudson for their papers.

Such electrocardiographic changes have been observed experimentally, but this seems to be the only example of coronary occlusion in the human being following

injury to the heart. The fact that there was an interval of some thirty-six hours between the time of injury and the development of the electrocardiographic changes carries a definite clinical application. During the period immediately following coronary closure the electrocardiogram may fail to show its characteristic signs of coronary closure and it may be only later that these signs appear. For early differential diagnosis between coronary closure and certain acute abdominal conditions the electrocardiogram, therefore, may not be of value. That is, a normal electrocardiogram does not necessarily exclude coronary closure if the electrocardiogram is taken early, within the first thirty-six hours of the accident.

I wish Dr. Elkin had told us something of his experience in suturing these wounds. The opportunity to suture a cardiac wound is given to few surgeons and Dr. Elkin and Dr. Phillips have had the experience of two cases. Sir Charles Ballance, discussing this subject, says that "the surgeon having this job in hand will take it all in the day's work, and just as he plunges his hand into the abdomen into a mass of blood, in a case of ruptured spleen or in a case of ruptured tubal gestation and seizes the bleeding spot, so he will now plunge his hand into the pericardium and seize the heart, and, by digital compression, control the hemorrhage, and proceed to suture the wound." The suture of a cardiac wound is not always so simple and there are certain points that may be helpful. Several years ago I described a method for the suture of cardiac wounds based on experience obtained in the laboratory. This method consisted first of placing a suture in the apex to steady the heart. This suture is held in the left hand. Both the heart and the hand move in unison while the index finger of the left hand is placed on the cardiac wound. This controls the bleeding. Sutures are then placed close to the wound on each side of the finger so that when these sutures are crossed and drawn taut the finger can be removed without hemorrhage. The wound is easily visible and now can be sutured carefully so that if a coronary vessel is adjacent to the wound it can be avoided in the suture. The control sutures and the apex suture are removed. Cardiac muscle is very friable and it is important not to try to plug the wound by inserting a finger into it. Another method for the suture of cardiac wound was described by Sauerbruch. This method consists in controlling the bleeding by compressing the venae cavae and the pulmonary veins between the fingers while the wound is being sutured. This method, while applicable in certain cases, carries with it a danger from anoxemia.

Dr. Hudson's case is a most remarkable one. He apparently achieved a good result. I personally had begun to feel that the Brauer operation should be abandoned. This feeling was based on the statistical results of the operation and also upon experiment. Some years ago Smith and Liggett collected 107 cases in which the Brauer operation had been performed. Some improvement was obtained in 84 per cent. Valvular disease was present in 29 per cent, leaving 71 per cent of the cases in which the circulatory disorder was due primarily to pericardial scar. This large group, uncomplicated by valvular disease, should have been amenable to complete or almost complete circulatory restoration. Only 29 per cent, however, were able to return to work—a result that obviously falls short of what pericardial surgery can offer.

From the experimental point of view I have been unable to produce any appreciable difference in the cardiac output of dogs by suturing the normal pericardium to the anterior thoracic wall on the left of the sternum. On the basis of such experiments it seems that the adhesions to the anterior thoracic wall do not in themselves play a major rôle in the development of circulatory failure. I do not mean to imply that such adhesions are of no significance but rather that they are only a part of a more extensive fibrosis over the pericardium and the surface of the heart.

The Brauer operation limits itself to an attack on the superficial terminus of the scar to the thoracic wall. It does not attack the central end of the scar on the surface of the heart. I have seen several cases in which the Brauer operation was done and in which nothing short of pericardiectomy should have been done.

Another point that I should like to make is that I believe the operation for the relief of pericardial scar should be carried out in one stage. The heart yields to the surgeon one major operative opportunity. If the circulation is only partially relieved of its impedimenta at operation it may fail to recover from the effect of pneumocardiac tamponade. This point may be expressed mathematically. Let the circulation be represented by 100 and the relief afforded by operation on the thoracic wall be represented by 20, so that the circulation is now represented by 120. The bump inflected upon the circulation by exposing the mediastinal structures to the pressure of the atmosphere measure about 35. The circulation is now reduced to 85, to a level that temporarily at least is not as efficient as the preoperative condition. Indeed, this level may be incompatible with life. As examples of this I may cite two cases; one operated upon by Dr. Elkin and the other by Dr. Cutler. These patients had little or no circulatory reserve and after the precordial ribs were removed the circulation gradually failed. The relief that the operation afforded was not sufficient to wipe out the effect of pneumocardiac tamponade. We look forward to a time in the future when all pericardial operations will be done in a negative chamber so that this complication will be avoided.

DR. DOCK.—I was very much interested in Dr. Hudson's work, for, after studying Dr. Beck's experimental paper last year, I still shared Dr. Hudson's belief that the main trouble was the adhesions fixing the heart to the surrounding viscera.

I don't believe the question can be settled by any single case or by any series of cases, but I might point out from the internists' standpoint that 27 per cent of the recoveries among decompensated rheumatic heart cases is really good for in studying effects of digitalis on rheumatic hearts, in only one case out of three does this drug have any effect. So I don't think surgeons should be discouraged if in more than half of the cases they fail to relieve cardiac failure. The whole question is one that will have to be solved by long observation of patients in whom cardiolysis is first performed, and then when this fails to relieve congestive heart failure, carrying out Dr. Beck's type of pericardial release to determine whether this will give better results.

It is possible for a small strip of scar tissue in the pericardium, constricting the coronary veins, to produce myocardial ischemia, and the freeing of the pericardium in such cases will be effective.

Dr. Elkin has reported a fine example of properly controlled and continued study of the effects of coronary occlusion.

The changes in the electrocardiograph following coronary occlusion are the theoretically understood from autopsy and animal experiments, but it is hard for internists to believe that the coronary artery can be completely occluded and yet changes in the electrocardiogram not ensue for many hours. When changes appear late there is always some discussion as to whether progressive thrombosis is going on. Dr. Elkin has furnished valid evidence that such need not be the case.

We hope that surgeons everywhere, in dealing with operations on the heart, will take advantage of their opportunities to study electrocardiographic changes in similar instances and also in cases when they have the heart exposed to study the form of the extrasystole produced by simple mechanical stimulation of the heart.

DR. BECK.—May I ask Dr. Elkin whether there were any changes in the Q-wave?

DR. ELKIN.—Yes.

PRESIDENT BUTLER.—Any further discussion? If there is no further discussion we will ask Dr. Elkin to close.

DR. ELKIN.—In answer to Dr. Beck's question, the measurements show a low Q, such as is found clinically in coronary occlusion.

I think it is unfortunate that we can't keep all the things in mind when an experiment such as suture of the heart is presented. We would like to know exactly what the venous pressure is in cases of actual tamponade as compared with the amount of blood that was in the heart at the time, which in future cases should certainly be determined.

I would like to emphasize again that the electrocardiogram is of very little value in differentiating between coronary occlusion and acute abdominal diseases. That is, it may show no change until several hours after the coronary occlusion may have occurred, in which case it may be too late to be of any value in diagnosis.

DR. HUDSON.—There is very little for me to say in closing, except that we were cognizant of the matters that Dr. Beck has brought up in his discussion at the time we planned this attack on this young man. But in going over the situation it seems to us that we had certain definite facts to face, and that was the fact that the series of cases which Dr. Dock and Dr. Beck have spoken of, tell us that we might get some relief from the type of operation we have performed. In addition, however, it seemed to us that certain of the deaths that had been reported might be accounted for by the fact that there had been too sudden a change in the intramediastinal and intrapleural pressure; and it seemed that it might be possible, by releasing the base of the heart first and gradually working toward the apex to overcome that difficulty and permit a gradual readjustment.

We had to bear in mind we were dealing with a patient who was not in very good condition, and for that reason we were inclined to temporize. And as I mentioned in closing, we may find it necessary later to go in and sever some adhesions, or even to remove a portion of the pericardium before we feel entirely satisfied with this young man and our results. But with the improvement he has made, we feel that at present, at least, he is a better risk for any future work than he was when we began.

# BRONCHIECTASIS*

*(To be published serially in four installments)*

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**Chapter I. Etiology and Pathology (Bacteriology discussed in Chapter II)**
**Chapter II. Clinical Features and Diagnosis**
**Chapter III. Treatment**
**Chapter IV. Treatment**
**Bibliography follows Chapter IV**

## I. ETIOLOGY AND PATHOLOGY

CAYOL in 1808 first directed Laennec's attention to dilatations of the bronchi and Laennec in 1819 described the diseased state of the bronchi now known as bronchiectasis.

### DEFINITION

Some authorities state that bronchiectasis consists of bronchial dilatations associated with bronchial and peribronchial changes which may produce thickenings and thinnings of the bronchial walls; others that it is a diseased state of the bronchi in which they become dilated and distorted producing dilatations and enlargements which may be single or multiple, local or general. The term bronchiectasis or bronchiectasia, however, merely means a dilated bronchus or dilated bronchi, and according to their form one may recognize saccular, cylindrical, fusiform and varicose dilatations.

### THEORIES CONCERNING MECHANISM OF PRODUCTION OF BRONCHIECTASIS

Mechanical pressure of stagnant secretions (Laennec).

Nutritional changes in the bronchial wall (Andral).

Forced inspiration (Raymond).

Neuromuscular mechanism (Stokes).

Intrapulmonic extrabronchial factors, "cirrhosis pulmonum" (Corrigan).

Relation of emphysema to bronchial dilatations (Rokitansky).

Predisposition (Hoffman).

Laennec regarded the bronchial dilatations as the primary anomaly and considered them to be a consequence of chronic and of repeated bronchitis (theory of mechanical pressure of stagnant secretions). Laennec described regular and irregular dilatations of the bronchi. These, he stated, could be associated with thickening of the

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affected bronchial wall and communicated with a system of cavities which resembled tuberculous cavities.

Andral stressed the importance of changes in the nutrition and texture of the bronchi in a communication published in 1824. He referred in particular to the consequences of hypertrophic changes in the bronchi (theory of nutritional changes in the bronchial wall: hypertrophy: atrophy).

Raymond discussed the question of forced inspiration in the production of bronchiectasis in a memoir entitled "Sur l'obliteration des bronches," published in 1834 (theory of forced inspiration). He suggested that in a given case all the air which was inspired could not be expired and was thus able to weaken a normal bronchus or to affect a bronchus which was already weakened. Mendelsohn also supported the inspiration theory advanced by Raymond, and pointed out that the pressure in the bronchi under ordinary circumstances does not exceed atmospheric pressure, but that with the glottis closed it can become one-seventh greater than atmospheric pressure. The work of Thornton and Pratt is interesting in this connection. MacCallum states that violent inspiratory distention of the lungs preparatory to coughing may be able to widen the bronchi if they are weak and if the act is constantly repeated. It is very doubtful whether coughing ever appreciably affects the bronchi of a normal lung.

Stokes spoke of paralysis of the circular muscle fibers of the bronchi with subsequent loss of ciliary action and atrophy of the musculature (theory of neuromuscular mechanism). He noted that retained secretions, changes in the bronchial mucous membrane and loss of contractility could produce dilatations of the bronchi.

Stokes described bronchial tubes which presented the appearance of fingers of a glove, others which resembled common bladder wrack (fucus vesiculosus) and a third type which looked like a large cavity or phthisical abscess. He also noted the association of bronchitis and bronchiectasis.

Corrigan in 1838 described the condition of cirrhosis of the lung and concluded that pathologic changes in the pulmonary parenchyma were responsible for the dilatations of the bronchi (theory which deals with intrapulmonic extrabronchial factors). It is obvious that the pulmonary parenchyma which surrounds a bronchial dilatation may be diseased and that the normal architecture may become completely obliterated as a result of fibrosis. Laennec would have us believe that such changes are always secondary; Corrigan, on the other hand, intimates that since the bronchi in a cirrhotic lung lie in a compressed lung, the dilatations must therefore of necessity be secondary.

Hamilton raises the question why it is that in cirrhosis of the liver or most other organs, dilatation of the tubular structures and blood

vessels does not take place as in cirrhosis of the lung. The explanation which he offers is as follows: The two pleural surfaces are normally practically united, the pleurisy which accompanies pulmonary cirrhosis renders this complete. The dilating force consists of the constricting bands which are attached to the pleura and bronchial wall. The pull is to the chest wall. The chest wall forms an arch with its concavity to the point of traction and will thus be affected less by the pull than a bronchus which has its convexity toward the point of traction. We may add that under most conditions it is easy for a force to exert a pull on a bronchus, but that it is said to be well nigh impossible to pull the two pleural surfaces apart.

Bronchi near the surface and borders of the lung and those in contact with areas of emphysema are in the opinion of Rokitansky the most likely ones to become dilated (theory which concerns relation of emphysema to bronchial dilatations).

Hasse and Rokitansky observed that bronchiectasis frequently followed upon chronic infective lesions such as bronchitis and tuberculosis; that when it followed upon the former the lesion developed in a relatively much shorter time; that late changes were always deep seated, parenchymal and that bronchiectasis which resulted from chronic bronchitis was of the cylindrical form. Engel did not concur with this latter remark.

Rokitansky remarked that whatever be the form under which bronchial dilatations appear, bronchitis must be regarded as a "primary" cause. Although this need not be so in every instance, certain it is, however, that it is frequently an associated finding.

Rillet and Barthez in 1843 drew attention to the fact that bronchiectasis could frequently be considered even when there was no evidence of chronic bronchitis. They observed bronchiectasis twenty-one days after the onset of an acute bronchitis.

Marfan says that the bronchopneumonia of influenza, measles, whooping cough and typhoid fever predisposes to bronchiectasis. Hoffman does not agree with this statement. He feels that a special malignancy in these diseases is necessary to bring about such consequences and adds that upon what this malignancy depends is not always clear (theory of predisposition).

Black summed up the situation more or less when he wrote as follows: "Hence occur saccular dilatations of the bronchi at the isolated points where the fibers of these tubes are embedded in one mass of organized exudation; or general dilatation when the whole extent of their fibrous tissue is involved and more particularly so when the above-mentioned condition and the complete or partial obliteration of other bronchi causes more air to be forced into such tubes than they are accustomed to receive in the healthy actions of the lung."

## BRONCHIECTASIS DURING CHILDHOOD

### CONGENITAL BRONCHIECTASIS

a. Universal

b. Telangiectacic

#### BRONCHIECTASIS DURING CHILDHOOD: CONGENITAL BRONCHIECTASIS

Bronchiectasis has often been called a disease of childhood and perhaps justly so. For even though the true congenital or atelectatic type of bronchiectasis may be of infrequent occurrence, still bronchiectasis is so frequently met with in children in whom the dilatations are so marked, and cough and expectoration so slight, that one feels that dry bronchiectatic dilatations may be present if not from birth, at least for many years before they attract attention.

#### CONGENITAL BRONCHIECTASIS: UNIVERSAL, TELANGIECTATIC

Congenital bronchiectasis is met with in two main forms. These are the so-called *universal* and *telangiectatic bronchiectasis*. They often fail to produce symptoms although cavities the size of a hen's egg may be present in the lungs of infants a few months old. Such cavities are sometimes found persisting in adults. These forms were described by Meyer (1859), Biermer (1860), Grawitz, Barlow (1880), Frühwald (1885), Heller, Kölliker (1886), Herxheimer (1887), Kaufman (1892), Stoerk (1897) and others. In the telangiectatic form the lung or part of it is found to be converted into a mass of cysts lined with high epithelium. The cysts may be quite empty and so suggest a pneumothorax pocket. Such cyst-like dilatations may rupture and are thus not infrequently the cause of a spontaneous pneumothorax. (Fig. 1, *a* and *b*, *c* and *d*, and *e*.) The cysts may fail to communicate with bronchi.

Universal bronchiectasis is a generalized form which is said to affect an entire bronchus by virtue of edematous degeneration. When cystic degeneration is present it usually is associated with bronchial stenosis which is due to either a true stricture or to the consequences of retained secretion and edema of the mucous membrane.

#### INTRAUTERINE INFLUENCES

It is perhaps not amiss to refer at this point to the fact that when a living fetus near full term is exposed within the uterus with the amniotic sac still unruptured, that it can readily be stimulated to make respiratory movements (Addison and How), and fetal movements (E. A. Graham). Graham showed that asphyxia of the fetus could be produced experimentally by (1) ligation of the uterine vessels, (2) occlusion of the trachea of the mother and (3) by direct inhalation of $CO_2$ by the mother. The fetal movements which resulted varied from those of attempts at respiration to more general muscular contractions

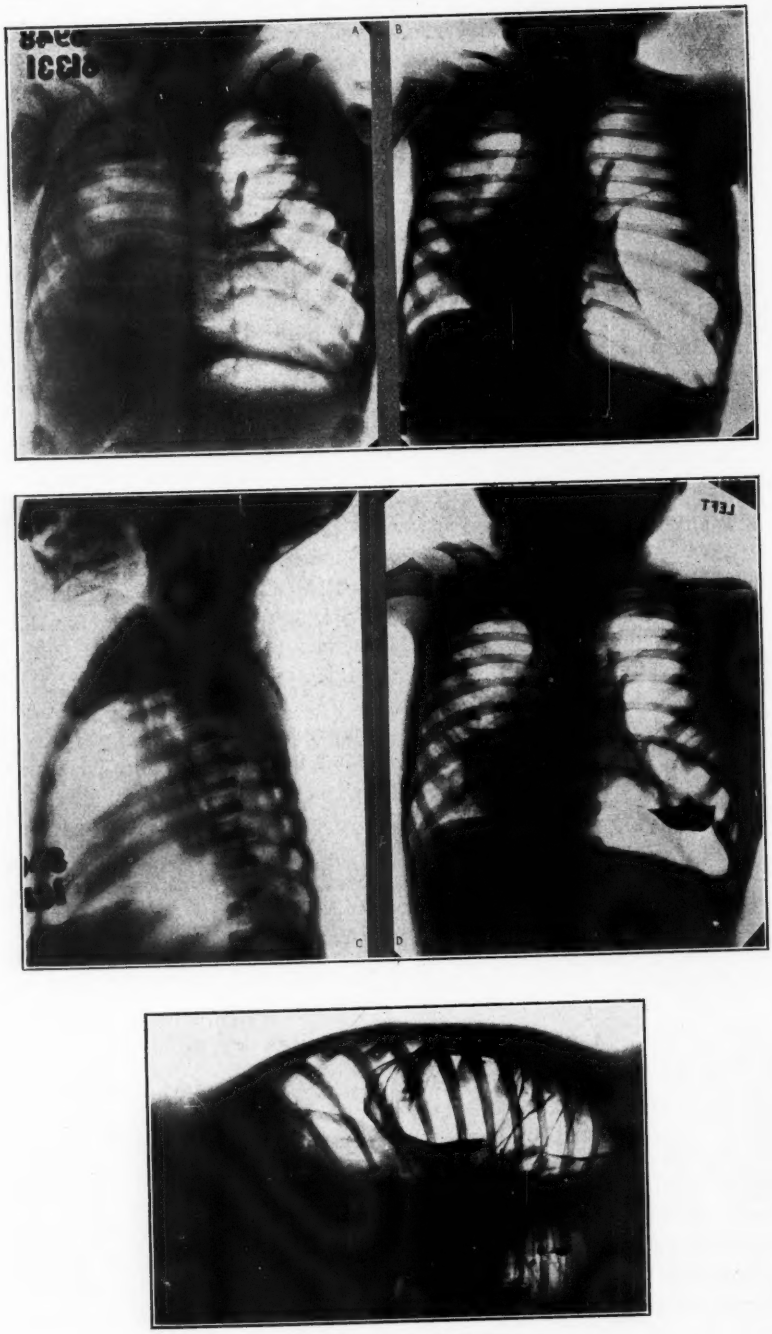


Fig. 1.—*A, B, C, D, E*

simulating the general convulsions of $CO_2$ poisoning seen in the adult. Geyls' experiments of injecting aniline blue into the amniotic sacs of fetuses are also interesting in this connection. During prenatal life future respiratory passages are filled with a liquid. Before breathing the lining epithelial cells of the alveoli are irregularly cuboidal with round nuclei. After respiration takes place the nuclei become spaced further apart, the cytoplasm drawn out and the cells appear very flat and thin. The blood vessels then become distended and more conspicuous. In this connection the question of disturbed circulation in the mother because of vein anomalies has been raised. It is to be concluded that as a result of such disturbances the alveoli do not fill and the bronchi dilate. The type of bronchiectasis which is said to result is the atelectatic type. As a result of intrauterine apnea, extrauterine asphyxia may develop (Runge). So, too, changes in the mother's circulation which affect the $CO_2$ of the mother's blood or a drop in blood pressure or an internal hemorrhage may all affect the placental circulation. Premature separation of the placenta, compression of the umbilical cord and increased intracranial pressure are other considerations.

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Fig. 1, *A* and *B*.—Bilateral cystic disease of the lungs; bronchiectasis. Patient referred by Dr. Croswell of Memphis.

The above film (*A*) was taken several days after the onset of an attack of acute dyspnea, cyanosis, spasmodic cough and fever in an eighteen-month-old child. This attack followed an acute upper respiratory tract infection. Before the child was seen by us, the mediastinal contents were found to be displaced to the right, and closed drainage of apparently the left pleural cavity, low down, was established at Memphis. Yet, in spite of the positive pressure, an effusion failed to develop. No improvement followed the above procedure and the tube was withdrawn after a few days. The skin wound healed over. A bronchoscopic examination at this time was negative. From then on the child obtained some temporary relief as a result of daily aspirations of over 1000 c.c. of air from the left upper chest performed by Dr. Croswell.

The films 1-*A* and *B* show the large cysts and what appears to be a pneumothorax pocket at the left base. Fig. 1 *B*, shows considerable clearing of the outer margin of the right lung. This may have been due to a pneumonic process. Past history of bronchopneumonia at five and thirteen months, dyspnea and cyanosis marked during both attacks, has had frequent colds since, and suppurative otitis media.)

Fig. 1, *C* and *D*.—Bilateral cystic disease of the lungs, bronchiectasis. Same patient as in Fig. 1-*A* and *B*.

Fig. 1-*C*. (Lateral view) A fluoroscopic examination at the Barnes Hospital on August 5, 1931, which was about twelve weeks after the onset of the child's illness, still revealed the fact that the mediastinal contents were displaced to the right. The left diaphragm showed paradox movement. This fact, together with the above, suggested that the air pocket above the left diaphragm was a tension pneumothorax. A needle was introduced high up in the left upper chest. The pressures were +20 and +18. Eight hundred c.c. of air were then withdrawn with the aid of a pneumothorax apparatus. The pressure then registered +6 and +4. The mediastinal contents had returned to their normal position. The child experienced great relief from this aspiration. Fig. 1-*D*. Six c.c. of lipiodol were then introduced through the same needle. The oil dropped to the bottom of one of the cysts of the left lung and under the fluoroscope one could see the oil run over both main bronchi into the opposite lung. The air pocket over the left diaphragm failed to be injected. The lipiodol injection demonstrated beyond doubt that the condition was one of bilateral congenital cystic disease of the lung. This type of cystic disease is often considered to be a form of bronchiectasis and is frequently associated with smaller dilatations and with acquired bronchiectasis. The latter develops as the result of repeated infections.

Fig. 1, *E*.—Bilateral cystic disease of the lungs; bronchiectasis. Same patient as in Fig. 1, *A*, *B*, *C*, and *D*.

This film was taken with the patient's left side up. A communication from Dr. Croswell on September 24, 1931, stated that the child had developed a left-sided pleural effusion and that the mediastinal contents had returned to their normal position following the aspiration of a small amount of fluid. Further aspirations of air then became unnecessary. The perforation in one of the cysts which was allowing the escape of air into the left pleural cavity had undoubtedly become sealed off.

Ewart, Wilson and others have been able to show that the lung of the child is just as complex in structure as that of the adult. Subsequent growth would appear to be dependent on an increase in the size of the alveoli without any addition to their number.

### DEVELOPMENTAL ANOMALIES

According to Hueter many of the so-called cases of congenital bronchiectasis are due to developmental anomalies in the bronchial tree. If the arrest in development is early, only large bronchi are formed

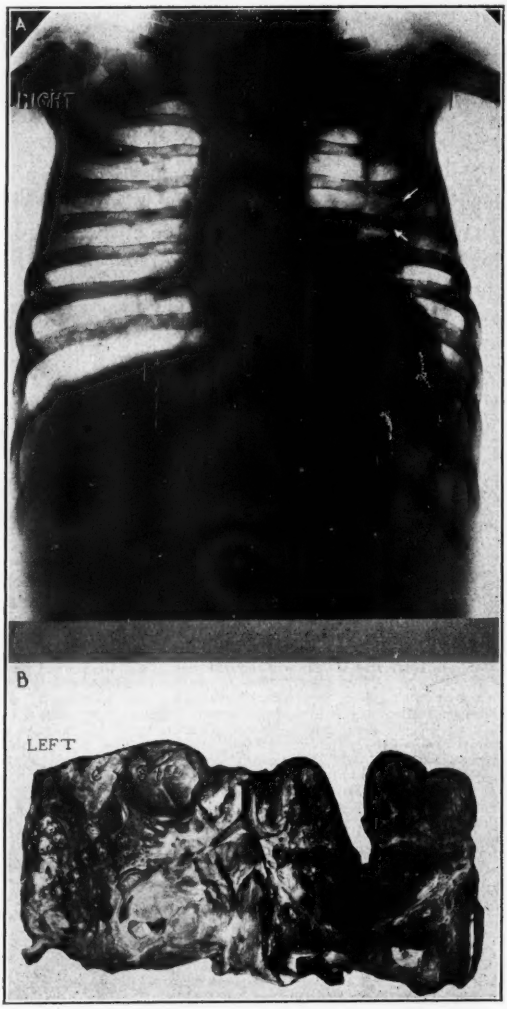


**Fig. 2.**

and the rest of the lung may be a sac. Some look upon atelectasis as a simple delay in development (Buchmann). Dilatations of the bronchi may also result from hypoplasia of the alveolar structure (Hondo).

Brauer and Wiese have repeatedly expressed the thought that the sum total of all anomalies which are met with in the bronchial tree play an insignificant rôle in the etiology of bronchiectasis.

### FETAL ADENOMA

The observation of marked epithelial proliferation in some instances of telangiectatic bronchiectasis led Stoerk to consider the possibility that this condition might be an expression of a true fetal adenoma of the lung. Löhlein has presented a cystic papillary (true) tumor which has aroused discussion regarding the relationship of such a tumor to the anomalies described by Grawitz and Stoerk.

### CONGENITAL CYSTIC DISEASE

Virchow described a case of bilateral "cystic" bronchiectasis in a cretin. He concluded that the cysts in this case were formed from dilated lymphatics and compared the condition to congenital macroglossia. Grawitz compared telangiectatic bronchiectasis to congenital cystic kidney and considered the process to be the result of hydropic dilatation of a main bronchus and its branches. Grawitz found goiter associated with this malformation.

Sauerbruch points out that upon a developmental basis it is conceivable that a bronchus may be "tied off" when the lung is being enveloped by pleura. If a large bronchus is so affected, then a large cyst of the lung results. If the middle-sized bronchi are involved, then smaller cysts will develop and, if this "tying off" occurs during the last intrauterine developmental period, then multiple small cysts or congenital bronchiectasis will be the result. A superimposed infection, measles, scarlet fever, whooping cough will make evident the under-

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Fig. 2.—Congenital cystic disease, left, and bilateral bronchiectasis. The above film concerns a one-year-old male who suffered an attack of pneumonia (January 3, 1931) which never completely cleared up. He was admitted to the St. Louis Children's Hospital on May 19, 1931. A mastoidectomy was performed on June 19, 1931. The child died the following day. There were many râles throughout both chests. The physical findings suggested an air pocket in the left upper chest. Clubbing was marked.

*A*. The roentgen-ray film taken after admission shows a large air pocket in the left upper chest. It would be most unusual for such an air pocket to be due to a cavity in the lung other than a cyst. Children rarely develop tuberculous or abscess cavities of such a size. An emphysematous bleb in a child rarely reaches this size, and if the large air pocket were to be considered as a spontaneous pneumothorax cavity, then the mediastinal contents should have been displaced to the opposite side. If this air pocket was a pleural pocket, in other words, an empyema cavity, then one should have expected a fluid level. The physical findings, clinical history and increased detail throughout both lungs, together with the clubbing, all suggested that one was dealing with congenital cystic disease and bronchiectasis.

The lungs (*B*) have been cut in two and opened out like the leaves of a book. Note the large smooth-lined cysts occupying the greater part of the left upper lobe. The cyst wall is slightly trabeculated. It does not seem to communicate with the main bronchus. There is extensive bronchiectasis throughout the remaining portions of both lungs, particularly the left. This bronchiectasis has many of the characteristics of congenital bronchiectasis but there are many inflammatory changes. The condition could well be an acquired bronchiectasis associated with a congenital cyst or congenital bronchiectasis with superimposed inflammatory changes. The latter is most likely the case. Note also a small empyema cavity at the left base.

lying condition and we will then have bronchiectasis as it is seen clinically (Fig. 2). Congenital and acquired bronchiectasis may coexist. Reference has already been made to the other forms of congenital bronchiectasis (Meyer, Grawitz) in this connection.

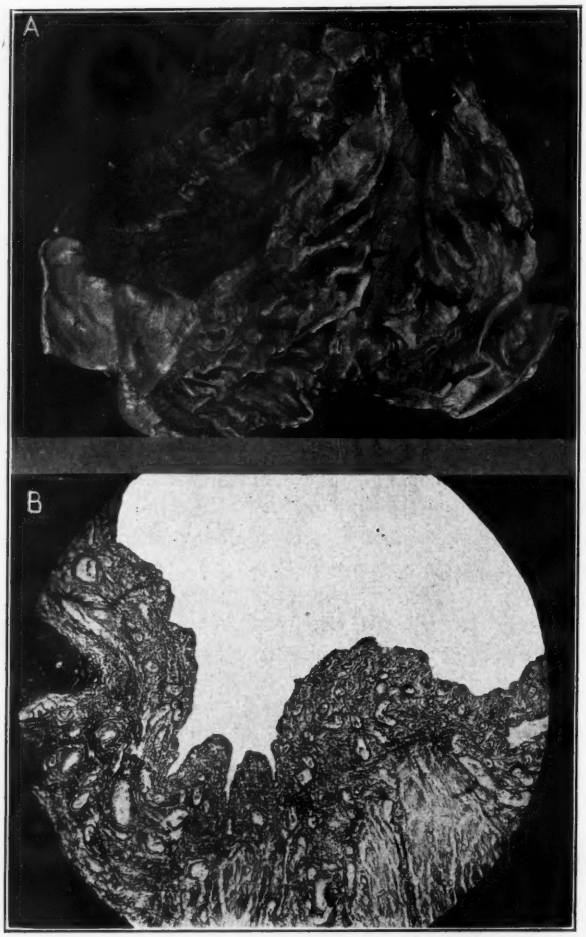


Fig. 3.—*A*, Congenital atelactatic bronchiectasis. B, Microphotograph, low magnification.

The alveoli in the atelectatic portion of the lung are markedly compressed and free from carbon pigment. The bronchi are very irregular and it is histologically difficult to differentiate adequately between their various layers. The various layers of the bronchi always remain more or less distinguishable in the acquired forms of bronchiectasis, even though there may be marked inflammatory changes.

## CONGENITAL PULMONARY EMPHYSEMA

Hueter has indicated that congenital cyst-like dilatations may be filled with either air or fluid and that it is important to differentiate

between widened alveoli and dilated bronchi. It has already been stated that bronchiectasis not infrequently develops at the site of emphysema.

### CONGENITAL ATELECTATIC BRONCHIECTASIS

Congenital atelectatic bronchiectasis is considered to be that widening of the bronchi which occurs in parts of the lung which have remained collapsed throughout life. Such a lobe or lobule may remain unpigmented and consists of a sponge of wide bronchi embedded in a firm tissue in which the alveoli can no longer be easily distinguished (Fig. 3). Such widening (Heller and Buchmann, 1911) may be the result of the tendency of a portion of the lung which is not taking its part in the normal movements of the thorax to dilate during inspiration. Ribbert and Feer state that the atelectatic lung parenchyma becomes invaded by fibrous tissue, becomes indurated and that air which should be spread over a large surface is able to act on the bronchi during inspiration and so weakens them. Feurstell and Heller speak of the hypertrophy of the various elements, while Herxheimer concludes that there need not be absence of pigment, that the epithelium is usually atypical and that there may be compensatory (emphysema) hypertrophy of the opposite lung. Koekert recently reported a case of fetal bronchiectasis occurring in a newborn child. The microscopic diagnosis in this instance was fetal bronchiectasis forming a cyst of the upper lobe of the left lung and atelectasis of both lower lobes. The cyst fluid was not examined for urea but it resembled amniotic fluid.

### PRIMARY ACQUIRED ATELECTASIS, SECONDARY BRONCHIECTASIS

Quite a number of years ago Hamilton made the following pregnant remark, "A fertile source of bronchiectasis in children is pulmonary collapse." And it is only now that full cognizance of this remark is being taken. When one but appreciates the number of foreign bodies which children aspirate into their bronchial trees, the fact that hilar glands may compress bronchi or even rupture into a bronchus, that in childhood the greater part of the lung is composed of bronchi and that upper respiratory infection has little or no difficulty in spreading down the entire bronchial tree which in a child is in a sense nothing but straight short tubes, then we begin to understand why many cases of bronchiectasis met with in the adult must have occurred in childhood. The course of the bronchi in some measure suggests why the air is unable to enter even collapsed peripheral portions of the lung when they are filled with secretions. The unaffected bronchi may undergo a compensatory expansion. Hoffman has stated that the bronchi also lose some of their resistance as a result of catarrh.

When mucus or secretion partially plug a bronchus, then each expiratory effort must compress a vesicular structure. The peripheral

bronchial tubes will in the course of time become dilated. Some degree of emphysema and bronchiectasis will develop later. It is well to remember that some of the blood vessels may be narrowed and obliterated during this process. It is perhaps safe to assume, from what has been said, that so-called postoperative massive atelectasis and patchy atelectasis play a part in the production of some cases of bronchiectasis.

### ABSENCE OF ANTHRACOTIC PIGMENT

It has often been considered that a criterion of congenital bronchiectasis with atelectasis is an absence of coal pigment in the affected portion of lung. But one cannot always depend upon this feature to make a diagnosis of congenital atelectasis for a foreign body swallowed during infancy may produce the same picture. It is, therefore, not always easy to differentiate between intra- and extrauterine atelectatic bronchiectasis.

### HERNIA OF THE LUNG AND BRONCHIECTASIS

Frühwald has reported an instance of intercostal lung hernia which was associated with fetal bronchiectasis.

### PNEUMONIA AND BRONCHIECTASIS

Marfan says that the bronchopneumonia of influenza, measles, whooping cough and typhoid fever predisposes to bronchiectasis. Hoffman does not agree with this statement in that he feels that a special malignancy in these diseases is necessary to bring about such consequences and adds that upon what this malignancy depends is not always clear. We are inclined to agree with Hoffman. Reference to this point will again be made elsewhere. Some other opinions concerning the association of pneumonia will here be made. Thus, Rapp felt that most bronchiectatic lesions followed upon a lobar pneumonia.

Dilatations of small bronchi are always accompanied by a certain amount of chronic pneumonia. When such is the case the origin of the bronchiectasis is always multiple (Hutinel).

Bezançon considers bronchial dilatations to be caused either by bronchopneumonia which leads to a disappearance of the fibroelastic tissue of the bronchi or to repeated attacks of bronchitis which as a result of coughing causes a distention of the bronchial walls. He notes, however, that many cases of bronchiectasis have a doubtful etiology, particularly those of an insidious progressive onset and without a clinical history of bronchopulmonary disease.

In discussing the development of bronchiectasis, Sauerbruch has pointed out that it may be a consequence of an inflammatory process in the lungs such as a chronic pneumonia. There are instances in which the development of bronchiectasis resembles compensatory em-

physema. In other words, bronchiectasis is a compensatory phenomenon. Sauerbruch favors the congenital type. In the congenital type no history of pneumonia, grippe or similar infections is forthcoming, but since childhood there has apparently been a chronic catarrhal condition in the bronchi. When such is the case, the pleura is never found to be adherent. Greenish blue areas which correspond to peripherally dilated bronchi are visible. Chronic inflammatory changes are confined to the bronchi. Sauerbruch states that 80 per cent of this type of bronchiectasis are to be observed in the left lower lobe. In the congenital form the contour and structure of the bronchial wall are very irregular and no sharp differentiation between the various layers can be made out. The various layers of the bronchi remain more or less distinguishable in the secondary forms of bronchiectasis, even though there may be marked inflammatory changes.

Lederer has described a series of cases of pneumonia in young infants and has shown that induration and shrinking of the pulmonary parenchyma may develop very rapidly. We have observed it develop in less than six weeks, and with it the formation of bronchial dilatations.

The etiologic factor in bronchiectasis is according to Lederer not measles or pertussis but the associated bronchitis and pneumonia. The condition may develop during the course of years and can be followed by reinfection, by disease of the tracheobronchial glands and by pulmonary induration. Lederer states that the development of adenopathy means that reinfection may start from them through lymphatics which pass from the glands to the bronchi. There are those who believe in the theory of retrograde infection from the glands to the lungs by way of the lymphatics. The evidence for this point of view is not entirely convincing. It would seem that the facts could be equally well explained if one assumed that the reinfection of the lungs from the glands occurred through the entrance of the organisms into the general circulation and from there into the pulmonary circulation.

### BRONCHIOLECTASIS

Loeschke has emphasized the fact that the severe inflammatory pulmonary processes in children primarily affect the preterminal bronchi and that ectasia of bronchi so affected results. In the adult the middle and large-sized bronchi are usually affected.

Bronchiolectasis in children with reference to the condition known as honeycombed lung has been discussed by Leach. He feels that from the clinical standpoint the condition is not tuberculosis because of the marked lowness of temperature, negative Calmette reaction and absence of tubercle bacilli. Concerning the histology of the condition he has noted that some of the cavities have no epithelial lining and that others have their walls lined with a layer of inflammatory lymph.

These cavities are bounded by collapsed alveoli which may show signs of considerable inflammation. The cavities were of three kinds:

a. Some dilated bronchioles or a true bronchiolectasis
b. Some due to dilatation of the atria and infundibula, hence atriectastes and infundibulectases with emphysema
c. Some are abscess cavities which have discharged their contents into a bronchiole and have become enlarged by a process of inflation.

In discussing the condition it is noted that some consider it to be acute bronchiectasis, others, cavity formation with destruction of the walls of the neighboring alveoli, constituting in reality a form of emphysema, and still others that it is a combination of the two above-mentioned conditions.

Vogt states that in chronic bronchiolectasis the predominant microorganism is the influenza bacillus.

Box has also made some observations on honeycomb lung and bronchiectasis in childhood. He has observed that the pleural surfaces more often than not are adherent and that superficial bullae project as small transparent blisters beneath the pleural envelope. The cavities may or may not communicate. Some investigators, he states, regard the condition as a consequence of broken down bronchopneumonic masses, others that they are abscess cavities or dilated bronchi, and still others, as we have already noted, that they are dilated infundibula. Giant cells occur in the walls of such cavities. Box considers the saccular bronchiectasis of adults but an advanced form of honeycomb lung. He remarks that the sputum of such patients is fetid, hemoptysis seldom occurs, and that one of the consequences may be mediastinal and subcutaneous emphysema.

### MEDIASTINAL SWELLINGS AND BRONCHIECTASIS

With the exception of enlarged mediastinal glands intrathoracic swellings are seldom responsible for bronchial dilatations in children.

### COMPRESSION OF THE LUNG AND BRONCHIECTASIS

Williams remarked that if compression by fluid in the pleural space were kept up for a long time in cases of pleuropneumonia that then certain areas of the lung would not expand and that the larger and middle-sized bronchi would not be obliterated but would take all the stress and strain. Brauer has never observed a single instance in which he felt that dilatations of bronchi had resulted from pneumothorax treatment which had been continued over a period of years.

### THORACIC DEFORMITIES AND BRONCHIECTASIS

Cohnheim first made the following observation concerning dilatations of bronchi which is not always sufficiently appreciated. It is to

the effect that whether the dilatations involve the smaller or larger bronchi, irrespective of their mode of origin or the manner in which they developed, that they can never occur in a closed thorax save at the expense of other parts which previously occupied the same space. In other words, when bronchi become ectatic some of the soft parts of the lung must give way. The alveoli usually give way and their destruction may precede the dilatation of the bronchi. This, of course, means that the thorax need not ordinarily undergo changes. Thus, Neisser in 1901 noted that congenital bronchiectasis is rarely accompanied by a deformity of the thorax. That this is not so in acquired types of bronchiectasis has been demonstrated by Orsos.

Softening of the ribs, weakness of the muscular elements, retention of secretion, in other words, weakening of respiratory efforts, may in some cases lead to bronchiectasis. The rachitic thorax perhaps predisposes to bronchiectasis in that when such children contract pneumonia, atelectatic areas are more likely to persist and become bronchiectatic. Meyer has shown that the chronic abdominal distention which many rachitic children suffer from may be responsible for disturbances in the pulmonary circulation. In discussing the infantile thorax of the rachitic child as a predisposing factor in bronchical infections Foote expressed the opinion that a child with rickets has the infantile or neonatal type of thorax. The thorax of the newborn infant is round in its outline when measured at the nipple. As childhood is reached it gradually becomes more elliptical. The thorax of a rachitic child may have all the characteristics of the chest of the newborn child. This potentially weakened thoracic cage may cause a lack of sufficient negative pressure in the pleura, this in time to incomplete alveolar distention. Rickets also predisposes to bronchotetany and this is also said to predispose to respiratory infections.

The thoracic cage may be quite normal in all forms of bronchiectasis. Skeletal changes which may be present are most frequently associated changes; rarely the cause of bronchiectasis.

### SYPHILIS AND BRONCHIECTASIS

Bronchiectasis in the fetus has been described by Kervity and others. Kervity demonstrated spirochetes in the bronchi of a thirty-six-hour-old child. He classified syphilitic bronchiectasis in the fetus and young child as follows:

1. Bronchiectasis with adenomatous formation
   (a) Adenomatous formation in the bronchi; epithelium changed to the cuboidal form or to high cylindrical form
2. Bronchiectasis, inflammatory
   (a) Bronchopneumonia of certain syphilitic origin
   (b) Bronchopneumonia of questionable syphilitic origin

According to Balzer and Grandhomme the syphilitic bronchopneumonia in children which causes bronchial dilatation is not common. They state that the most marked changes are in the intralobar bronchi, and that the lung increases in size because of cystic dilatations. This fact is not always acceptable for the cysts need not communicate with one another or with bronchi. The cyst fluid is usually clear. The pulmonary substance is reduced in amount and the intralobar bronchi are not always recognizable. Some areas seem to consist of dilated alveoli only. The process throughout is interstitial but the adventitial coat of the bronchi may be infiltrated. Balzer and Grandhomme believe that the diffuse and telangiectatic types of bronchiectasis (Grawitz) resemble the cases described by them.

It is interesting to note that Valentine states that the lungs have completed their development by the sixth week of intrauterine life, for there are some who believe that pulmonary syphilis of the fetus does not essentially differ from that of the adult.

Sandoz is of the opinion that congenital syphilis might be responsible for the production of bronchiectasis because he observed the condition in twin sisters. He concluded that as the changes had undoubtedly taken place in utero, syphilis afforded a likely explanation. We have observed bronchiectasis in twins and the occurrence of a bronchiectasis in three sisters has recently been reported. Siemens in his work on the pathology of twins makes no mention of this condition, however, so that it is probably not of frequent occurrence. A predisposition to bronchiectasis, in the sense of the constitutional pathologist, does not seem likely.

Connor has made an analysis of 128 cases of syphilitic stenosis of the bronchi and has reported an additional case. Among 97 postmortems dilatations of one bronchus or its branches occurred nine times. Cylindrical bronchiectasis was observed six times. We have observed such cases. (Fig. 4.)

Steinheil has described a form of syphilis of the lung associated with tracheobronchial syphilis which produces dilatations of the smaller bronchi and which resembles tuberculosis. It is doubtful if the latter changes are specific. Phthisis luetica has of course been referred to by many. Soft pleural caseous syphilitic nodules have been described by Worm, but it is admitted that part of the reaction noted about the nodule is unspecific.

The view held by some that many bronchiectatic dilatations are in a sense aneurysmal dilatations still requires further proof.

It is worthy of note that Faure has spoken of syphilitic pachybronchitis and that de Jong is of the opinion that gummata of the lung may lead to necrosis, sclerosis or bronchopneumonia which may be clinically expressed by bronchiectasis.

The formation of new smooth muscle fibers which may be observed in syphilis of the lung is unfortunately not a constant finding. The same can be said for maldevelopments of the bronchial tree and an arrest in the growth of elastic tissue which have also been attributed to congenital syphilis.

De Jong considers that the finding of Spirocheta pallida in the lungs of a newborn is but an expression of a septicemia. Windholz states that the response to salvarsan cannot be considered to be specific

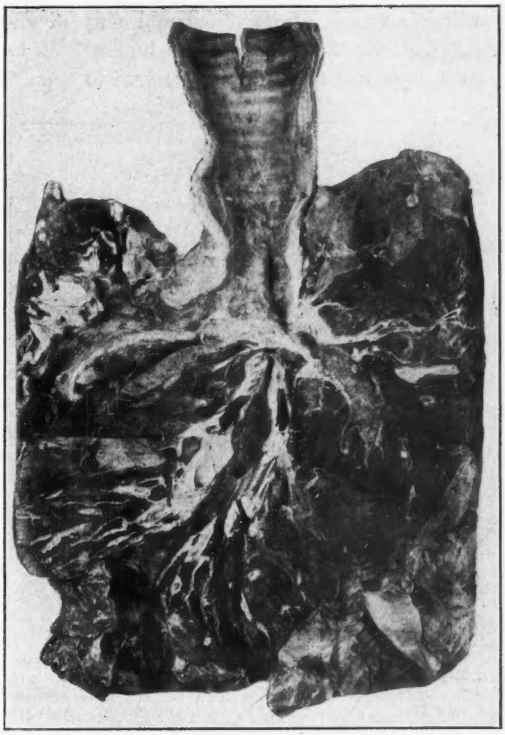

Fig. 4.—Bronchiectasis due to syphilitic bronchostenosis.

since bronchiectasis, fusospirochetal disease and other conditions respond to salvarsan. He adds why not state, as did Ornstein, that a patient with syphilis and lung signs and symptoms has syphilis with pulmonary symptoms, rather than designate the pulmonary symptoms as syphilis of the lung. We heartily agree with this statement. Windholz reports what he considers to be a true example of syphilis of the lung in a male, seventy-three years old. The right middle and lower lobes were involved. The bronchi showed obliterative changes and

peribronchitis. There was no evidence of tuberculosis; the blood Wassermann reaction was four-plus.

It must be admitted that syphilis of the lung is diagnosed clinically more often than pathologically and that syphilitic interstitial changes cannot be readily recognized microscopically. One must resort to animal inoculations in every case to exclude the possibility of tuberculosis.

In our investigations we have failed to discover any definite association between pulmonary syphilis and bronchiectasis. In but a few questionable instances could bronchial dilatations be attributed to syphilis. Syphilitic stenosis of main bronchi may produce an associated bronchiectasis. We have observed such cases. In two instances the bronchiectasis appeared to be of the congenital type (Fig. 5).

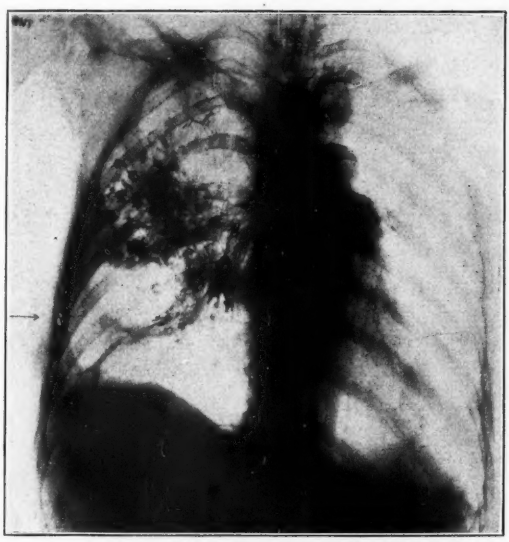

Fig. 5.—Saccular bronchiectasis, syphilitic in origin (?).

Large saccular bronchiectatic dilatations in a female, aged fifty-five years. The patient concerned had had syphilis for a great many years. She showed a typical syphilitic skin lesion, a saddle nose, and a perforated nasal septum and palate. Her blood Wassermann and Kahn reactions were repeatedly 4+. The complaints referable to her lung were rather mild cough and expectoration. She had for many years been treated for tuberculosis. This patient came to necropsy. The dilatations noted in the above figure were found to be rather large smooth-lined cavities which communicated with bronchi. This fact had already been suggested by the lipiodol injection since the oil had failed to enter the alveoli. Both lungs were covered by greatly thickened pleurae. There was no evidence of tuberculosis.

Dr. E. Burns who performed the necropsy in this case reported that numerous sections taken from the lungs showed almost identical lesions. There was diffuse fibrosis which took the form of large bands of connective tissue which occupied areas of lung substance which was devoid of alveoli. This connective tissue was extensively infiltrated with deposits of carbon pigment. There were also diffuse and localized areas of lymphocytic accumulations. The walls of the bronchi showed some degree of destruction but the epithelium was intact for the most part. The larger blood vessels throughout were found to be thick walled and their lumens narrowed. The aorta was normal.

The arrow indicates a localized pneumothorax which so compressed the corresponding portion of the lung as to prevent its injection.

Instructive works on the relationship of syphilis to diseases of the lungs and bronchi are those by Karshner and Karshner, Krohn, Brandenburg and Tanaka.

### BRONCHIECTASIS IN THE ADULT

Factors at play:

| | |
|---|---|
| a. Intrabronchial | Foreign bodies, bronchial stones (as a consequence, stricture), bronchial tumors (benign and malignant); (added factors, cough, forced inspiration, infections). |
| b. Bronchial | Mucous membrane changes (atrophy, proliferation, metaplasia, ulcer formation with healing by scar); bronchitis (catarrh); loss of elastic tissue; changes in muscle and cartilage; hypersecretion (overstimulation of glands, loss of contractility and sensibility; strictures (syphilis, etc.); vascular changes, fusospirochetal disease. |
| c. Peribronchial | Diseased and enlarged glands. Adhesions (mediastinal, etc.); tumors and enlarged intrathoracic organs, aneurysms. |
| d. Pulmonary | Pneumonia (broncho, lobar, interstitial); "pulmonary fibrosis"; tuberculosis, abscess of lung, tumor, anthracosis, Potter's lung, etc.; fungus diseases; emphysema. |
| e. Pleural | Bronchopleural fistula, adhesions, effusions. |
| f. Referable to thoracic cage | Deformities (rickets, predisposition to respiratory disease; weakening of thoracic force). |

### INTRABRONCHIAL FACTORS

Intrabronchial factors include foreign bodies, bronchial stones, benign and malignant tumors and mucus plugs. Reference has already been made to the latter.

Added factors irrespective of whether the cause lies inside or outside the bronchus are, of course, cough, forced inspiration, infection, presence of secretions and granulation tissue.

### FOREIGN BODIES IN THE LUNG AND BRONCHIECTASIS

That pure mechanical factors play as important a rôle in the production of bronchiectasis as they do in renal infection was noted by Rokitansky in 1860 and by Cohnheim in 1880. Paul Claisse many years ago wrote that prolonged sojourn of a foreign body in a bronchus might result in progressive dilatation of that bronchus. More particular reference to this association will be made when the treatment of bronchiectasis due to foreign bodies is discussed. It is well to note at this point, however, that the dispersion of fragments of bone or foreign particles throughout the lung substances (war and civil injuries) may be responsible for the production of very stubborn bronchiectasis. It is obvious that in such cases the origin of the bronchiectasis is multiple. It is in many respects analogous to the bronchiectasis caused by bronchial stones.

BRONCHOLITHIASIS AND BRONCHIECTASIS

Stokes, Rokitansky and others have stated that they had never observed a single instance of pulmonary calculi without corresponding dilatations of the bronchi (Fig. 6). They were in all probability referring particularly to broncholiths and not to pneumoliths.

Six of ten patients whom we observed some time ago with broncholithiasis showed definite bronchiectasis. Many more have been ob-

Fig. 6.—Relatively early cylindrical bronchiectasis of the left lung due to bronchial stones. The arrow indicates a stone in situ. Neither the history nor histological findings throw any light as to the cause of the associated previously unrecognized empyema. The pulmonary parenchyma is well preserved throughout.

served since. In one of the six there was also an associated abscess of the lung. In three others a diagnosis of abscess of the lung had been made. In such cases an attempt to prove the presence of an associated bronchiectasis should always be made. When there is expectoration it is wise to assume that there is some suppuration present. If for any reason a stone is found which is known to be or to have come from a bronchus, then one can assume that the case is one of bronchi-

ectasis; but if the stone is known to be in the pulmonary parenchyma, then there must be an associated lung abscess. In any case, however, it is extremely difficult to be certain whether the stone is in a bronchus or in the pulmonary parenchyma. Both may be present at the same time.

The origin of bronchopulmonary lithiasis has in some instances been ascribed to bronchial dilatations. It is well known that bronchial stones may form in bronchiectatic cavities. Metastatic calcinosis is also another consideration in this connection.

It has been said that an associated active pulmonary tuberculosis is present when pure anthracosis is complicated by broncholithiasis.

## BENIGN AND MALIGNANT TUMORS OF THE BRONCHI AND LUNG, AND BRONCHIECTASIS

The relationship of tumors of the lung to the development of bronchiectasis is obvious, but it is nevertheless worthy of some discussion. Biermer was among the first to demonstrate the frequent association of lung tumors and bronchiectasis. He considered the latter to be a late manifestation.

In the nodular form of primary carcinoma of the lung the bronchiectasis may be the result of nonspecific inflammatory changes while in other forms it may result from infiltration of the bronchial wall by tumor tissue. All forms of pulmonary carcinoma are likely to produce bronchiectasis, bronchiectatic cavities and atelectasis. Such cavities often empty themselves by sudden outbursts.

In a review of 374 primary carcinomas of the lung Adler makes reference to the presence of an extensive bronchiectasis in 36 instances, while in 90 cases of sarcoma (?) of the lung the pathological notes make mention of bronchiectasis in but five instances. In the former the tumor frequently completely obstructed the lumen of a main bronchus either from within or from without.

Hicks has reported a case of bronchiectasis which was due to a malignant metaplastic pedunculated intrabronchial tumor. Such cases are rare.

It becomes obvious that if a bronchial tumor suddenly occludes the lumen of the bronchus that the corresponding portion of lung tissue will become atelectatic. If, on the other hand, the tumor grows gradually, then it will be possible for dilatations to develop beyond the site of the tumor, and, if there is an associated bronchitis with cough and expectoration, then these dilatations will become clinically demonstrable. With the further growth of the tumor and its invasion of the lumen of the bronchus, secretions will become more stagnant and the dilatations more marked; for the size of the dilatations is sometimes an expression of the amount of the secretion which is

retained. And it is not unusual for a tumor of the lung to be associated with a pure abscess of the lung although it is appreciated that the carcinoma itself may undergo degenerative changes and form an abscess (Graham). Lenk notes that cylindrical bronchiectasis may simulate the so-called lymphangitis carcinomatosa.

The dilatations may be due to metastases (Fig. 7). Such metastases involve the local regionary glands and narrow the lumen of a bronchus. In so doing they may produce a relative dilatation of the bronchus in front of the point of narrowing and dilatations of the smaller bronchi beyond this point. In addition metastases to the glands at the bifurcation may so alter the carina and course of the main bronchus as to produce narrowing and retention of secretion.

There is always a nonspecific inflammation which goes hand in hand with certain tumors of the lung. This nonspecific inflammation may be responsible for repeated attacks of fibrosis and so dilatations of the bronchi may develop purely upon this basis. In addition, certain tumors of a scirrhous nature produce much fibrosis and shrinking of the affected lobe so that it may become so small as to occupy part of the hilus on the corresponding side. In such instances the shrunken lobe rotates contra-clockwise and comes to lie paramediastinally. Such a fibrotic lobe may contain dilatations. We have had the opportunity of observing this phenomenon. The remaining lobe undergoes compensatory emphysema and occupies the space previously occupied by the fibrotic lobe.

One cannot conclude these remarks without stating that the frequent association of carcinoma of the lung and bronchiectasis rarely leads to clinical confusion. By that is meant that the experienced seldom fail to appreciate a case of bronchiectasis due to a tumor of the lung. The chief reason for this clinical appreciation lies in the fact that almost all patients with carcinoma of the lung present themselves for examination when they are suffering from atelectasis or bronchiectasis or other late consequences. Both of these above mentioned late effects are among the first to attract attention and make the nature of the underlying condition apparent in such cases.

### METAPLASIA AND BRONCHIECTASIS

Concerning the rôle of metaplasia of the bronchial epithelium in the production of bronchiectasis the following may be said: Kawamura produced metaplasia of the bronchial epithelium experimentally by irritants while Teutschlaender noted similar changes in the lungs of rats which died from bronchopneumonia and in the lungs of a child who died from measles. Goldzieher found such changes in the larger bronchi of a patient who died from diphtheria. Askanazy could demonstrate metaplasia in the lower respiratory passages in 38 out of 90

cases of epidemic influenza and in two instances of typical pneumonia. We have also observed such changes in association with bronchiectasis. In a previous communication Baló and Ballon suggested that perhaps the proliferative changes noted in Vitamin A deficiency (Wolbach, Goldblatt and Benischek) were but phases of metaplasia. Be this as it may, sufficient evidence has as yet not been forthcoming to suggest that metaplasia or proliferative changes of the bronchial epithelium are to any great extent responsible for the production of bronchiectasis, even when such changes are to be noted in the terminal smaller bronchi. They may, however, be responsible for some in-

Fig. 7.—Bronchiectasis due to partial bronchial stenosis caused by metastases from a carcinoma of the prostate.

stances of alveolarectasis and possibly for the formation of small cysts (caverniculae) in that they may further narrow the partially obliterated lumen of a bronchiole and cause retention of secretions.

### DEVELOPMENT OF CARCINOMA IN BRONCHIECTATIC DILATATIONS

That bronchiectasis is a frequent consequence of tumor of the lung is well known. But few instances of tumor formation in a bronchiectatic dilatation have, however, been recorded. Siegmund has described three instances. Two of these occurred in chronic pneumonically indurated lungs, the other in an apical scar, probably tubercu-

lous. It is appreciated that carcinomas not infrequently develop in tuberculous cavities. In two of Siegmund's cases the bronchiectasis was undoubtedly primary. The point of origin of the tumor could apparently be clearly and definitely discerned. In all three instances the bronchial epithelium showed definite changes. The cylindrical epithelium had become low cuboidal epithelium in two instances. The character of these cells could be well observed in the metastases.

### BRONCHOSTENOSIS AND BRONCHIECTASIS

Rokitansky compares the formation of certain types of bronchiectasis resulting from bronchial stenosis to that of pyonephrosis resulting from a ureteral stricture. Bronchial stenosis is frequently due to foreign bodies. They more commonly lodge in the nonbranched extra- and intrapulmonary portions of the bronchial tree and include those accidentally drawn through the larynx and true bronchial stones. These foreign bodies may be expelled leaving decubital ulcers which heal by scar formation. The resultant effects are naturally dependent upon the extent of the bronchial occlusion, and, when it is incomplete, then a bronchiectatic condition develops.

Bronchostenosis may thus be due to factors (1) within the lumen of the bronchus or (2) outside the bronchus. Mediastinal tumors, enlarged glands, etc., may be responsible for external compression while tumors, syphilitic and other scars, decubital ulcers, calculi, caseous material and fibrin plugs (fibrinous bronchitis) may all cause partial or complete obstruction of the bronchial lumen.

### BRONCHITIS: HYPERSECRETION AND BRONCHIECTASIS

In bronchitis the inflammation regularly involves the trachea, larger and medium-sized bronchi, less frequently the smaller bronchi. The condition is usually bilateral, but when local factors are at play this need not be the case. The whole process is a congestive one, may be superficial and limited to the mucous membrane. The walls of the smaller bronchi may remain unchanged even when they are involved by inflammation and when full of pus. Lipiodol injections, postmortem specimens, have verified these observations. The injection picture is normal; so too in putrid bronchitis unless secretions and granulations have plugged smaller bronchi and have resulted in areas of atelectasis. When there is doubt as to the involvement of the smaller bronchi, usually only aspiration of all secretions before injecting lipiodol will permit of a positive diagnosis of bronchiectasis or enable one to exclude it. In such cases as in chronic catarrhal bronchitis when one examines the bronchi after death, one is often struck by the want of proportion between the symptoms and the lesion. The same bronchi which during life were constantly pouring out large

quantities of inflammatory products may after death show but few changes from the normal. In other instances the bronchial walls may be trabeculated, the epithelial covering deformed, because of newly formed cells in the deeper layers. The connective tissue stroma of the bronchi and arteries may be thickened and infiltrated with inflammatory cells while the muscular coats may be thickened or thinned. There may be cylindrical dilatations of one or more bronchi. A similar condition may exist in chronic croupous bronchitis. In chronic bronchitis the mucosa and submucosa may show very considerable swelling, the vessels considerable dilatation. The mucous glands may show a condition of chronic catarrh and atrophy. Some of them may become cystic as a result of obstruction to their ducts.

Although it is undoubtedly true that thick mucoid secretions may exert a valvular action in the bronchi preventing the easy exit of air or that during certain phases of respiration a weakened tube may be kinked, still it does not seem likely that this is often the sole cause of bronchiectasis. The larger tubes appear rigid, less flexible, and when this is the case the smaller bronchi are practically always involved. The secretions below the level noted in the injection picture are difficult to expel. When such terminal dilatations are aspirated, then lipiodol may sink down into them after several days and may persist in them for a considerable time. In doubtful cases the patient should, therefore, be observed several days after the injection. It cannot be repeated too often that putrid bronchitis without bronchiectasis is of relatively frequent occurrence. Such patients may expectorate fully as much as those suffering from bronchiectasis.

Small dilatations ("beading," caverniculae) usually occur fairly early so that it is reasonable to expect that in many cases of bronchiectasis the original lesion is one of bronchiolitis or putrid bronchitis. In many cases bronchiectasis must be considered not only as a disease of the bronchi but also of the pulmonary parenchyma. This is not the case early, especially when the cause is chronic or repeated attacks of bronchitis.

It is possible for bronchial dilatations to become dry, that is, hypersecretion ceases and only returns when a fresh infection develops It is thus erroneous to believe, as Biermer pointed out, that such dilatations have always been dry; for there are undoubtedly certain cases of atropic bronchiectasis in which hypersecretion or a superimposed bronchitis makes the underlying lesions manifest. In such cases the pulmonary parenchyma may be comparatively free. What in many instances causes or is responsible for the atrophic condition of the bronchus, the laxness of the bronchial wall, and change in the tonus of the bronchial musculature, is often quite difficult to tell.

Can hypersecretion lead to bronchiectasis? We have observed several patients with asthmatic bronchitis who developed bronchiectasis and in whom this was perhaps the case. This need not be the case and is perhaps the exception rather than the rule. And it is well to remember that putrid bronchitis, a condition in which the bronchi may be drowned with pus, may persist for years without producing any bronchiectasis, even though the patient may have a chronic cough. So, too, not every chronic catarrh or emphysema, the result of cough, need lead to the formation of bronchial dilatations. It must be borne in mind, however, when marked changes in the pulmonary parenchyma are associated with capillary bronchitis and an accumulation of secretions in the terminal bronchial tubes, that cough may then produce bronchial dilatations which might otherwise not have resulted.

A bronchial mucous membrane may be swollen and not hyperemic. The reverse may also occur, while both changes may of course be present at the same time. If there is marked overfilling of the capillaries, then they become elongated, tortuous and form papillary excrescences which still remain covered by mucous membrane. They may form small aneurysms (Hanot and Gilbert). Hypersecretion may then be marked and it is easy to understand how under such conditions the formation of small ulcers may cause bleeding. Particles of lime may be found in the epithelium and superficial ulcerations may develop as a result of them. Such ulcerations may be covered with yellowish offensive exudate and heal by cicatrices.

The amount of catarrh is not always proportional to the dilatation which may be present, nor do changes in the musculature of the bronchi form any index of the degree of bronchial dilatation present. Loss of muscle elements without reduction of elastic tissue has been described by Trojanowsky. Atrophic changes and hypertrophic proliferative changes in the cartilage of the bronchi have also been described.

Virchow believed that it was not always possible to differentiate between ordinary cavities in the lung and bronchiectatic cavities and W. Fisher has rightly pointed out that it is only by the presence or absence of bronchial mucous membrane that one can determine if cavities are bronchiectatic or not.

Gairdner concluded that almost all so-called bronchial dilatations, particularly those presenting the abrupt sacculated character, were the result of ulcerative excavations of the lung which communicated with a bronchus.

According to Socoloff perichondritis is by itself seldom a cause of bronchiectasis. Connective tissue and muscle change in the bronchi may on rare occasions cause the development of bronchiectasis.

Biermer made sharper differentiations between the various types of bronchiectasis. Thus, he referred to ectasia of the bronchi with catarrhal swelling which would no longer be evident when the catarrh disappeared; to dilatations of the bronchi with chronic catarrh, hypertrophy and thickening of the bronchial mucous membrane, vessels, glands, cartilage and muscles. He also spoke of an ectasia of the bronchi in which the walls of the bronchi become thin, and atrophic and from which saccular dilatations develop. A fourth form to which Biermer referred is the so-called mixed or trabecular form in which there is degeneration of the musculature and submucosal elements with practically complete preservation of the mucous membrane. Tro-

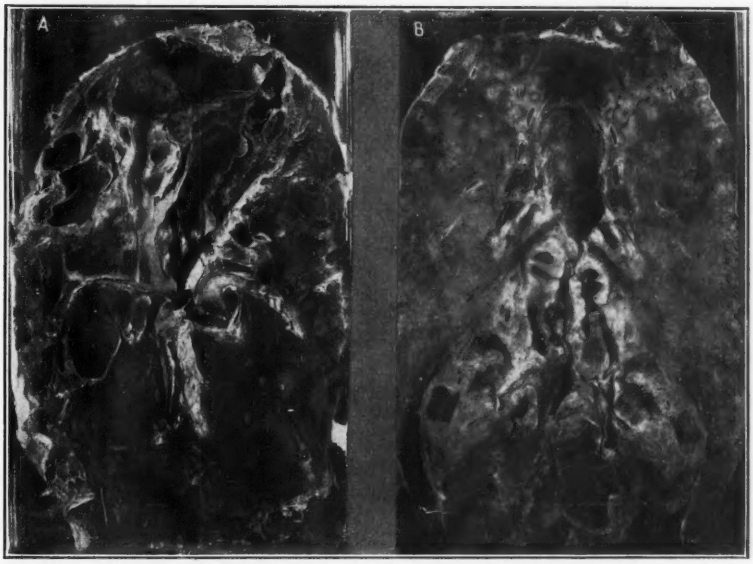


Fig. 8.—*A*, Saccular and cylindrical bronchiectasis. *B*, Cylindrical and sclerotic bronchiectasis for the most part confined to the lower portion of the lung. Such a lesion might roentgenologically appear as an area of paravertebral bronchiectasis and atelectasis. The remainder of the lung shows acute and chronic pneumonia and histologically much pulmonary sclerosis. The extensive infiltration by scar tissue is well indicated by the smoothness of the cut surface of the lung. One is dealing here with a form of Corrigan's cirrhosis pulmonum.

janowsky noted that the mucous membranes of the bronchi are in many cases of bronchiectasis not affected at all. Ballon made similar observations in a series of 75 specimens (Figs. 8 and 9).

Schneider has recently reported 215 cases of bronchiectasis which occurred over a period of twenty-one years (1906 to 1927, Aschoff). He speaks of but two forms, (1) cylindrical or diffuse, and (2) saccular or circumscribed. The two forms were combined in 157 instances. The lower lobes were affected in over 80 per cent. Schneider described the histologic appearance of the lesions as follows:

1. Pure atrophic bronchiectasis. In this form a bronchus is dilated in its entire extent down to the pleura. It may fail to show any acute narrowings at any point. This type is a form of the so-called cylindrical bronchiectasis (see Fig. 6). This form of bronchiectasis may also be combined with other types of bronchiectasis (see Fig. 8-*A*),

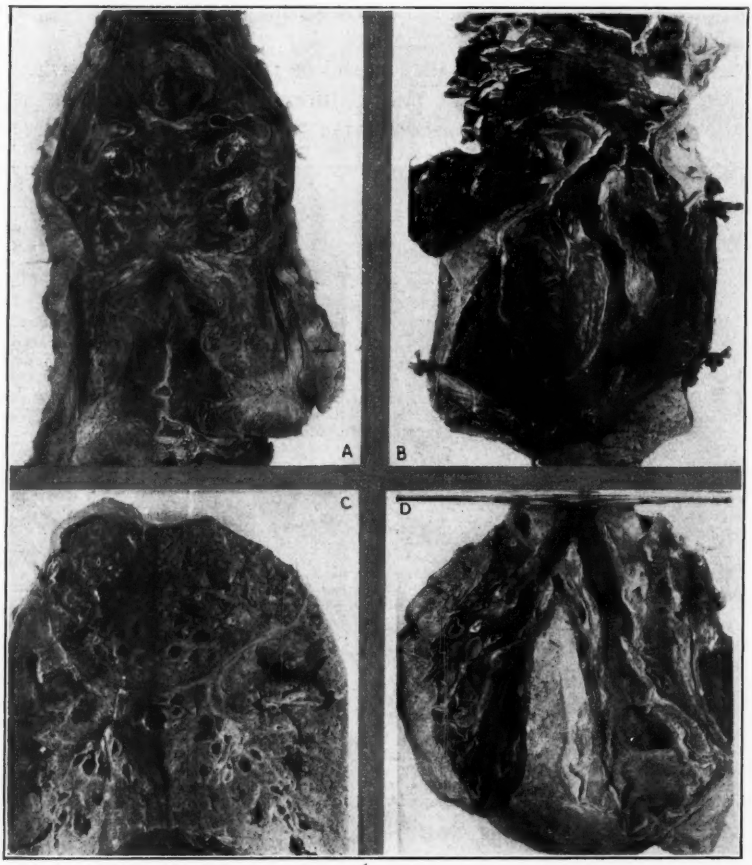


Fig. 9.—*A*, Cylindrical bronchiectasis. Note the rigid walls about some of the smaller cavities, and the rather large cavity at the base. The arrow indicates the remains of an encapsulated empyema. *B*, Cylindrical bronchiectasis. *C*, Sclerotic bronchiectasis in a tuberculous lung. *D*, Cylindrical bronchiectasis which has gone on to saccular formation in a lung which is otherwise rather well preserved.

and may be but an early stage of saccular bronchiectasis (see Fig. 9-*D*). Cylindrical bronchiectasis may also go on to the so-called sclerotic form which has been illustrated in Fig. 10-*A* in which case the sclerotic form of bronchiectasis is associated with the cylindrical form and with marked pulmonary induration. Fig. 10-*B* is an exam-

ple of sclerotic and cylindrical bronchiectasis which occurred in a tuberculous lung.

According to Schneider in a pure atrophic bronchiectasis the epithelium may be practically normal, the elastic tissue torn, the cartilage destroyed. The bronchial musculature shows no evidence of hypertrophy. The bronchial walls are free from cellular infiltration. There is no increase in connective tissue. The mucous membranes in the smaller bronchi may be normal and their musculature hypertrophic.

2. Atrophic type of bronchiectasis with inflammatory changes in the bronchial wall. Inflammatory changes are noted more particularly in the subepithelial layers extending to the muscle layers. Not infrequently in such instances the lung shows indurative changes (Fig. 11).

3. Sclerosing type of bronchiectasis. In this type the bronchi are sclerotic (see Figs. 9-*C* and 10).

4. Saccular bronchiectasis. (See Figs. 8-*A*, 9-*B*, 9-*D*, and 10-*A*.)
   a. Types in which the bronchiectatic cavities have retained their elastic tissue.
   b. Types with irregular rigid cavities (see Fig. 9-*A*).

It is worthy of note that in Schneider's series the pleura was adherent in ninety-one instances of cylindrical bronchiectasis (without adhesions, sixty-six) and in forty-two instances of saccular bronchiectasis (without adhesions, sixteen). In our experience the finding of an adherent pleura in bronchiectasis is not as frequent an occurrence as was formerly believed.

An attempt will be made later to correlate the lipiodol injection picture with the underlying pathologic lesion (Fig. 12).

### FUSOSPIROCHETAL DISEASE AND BRONCHIECTASIS

David T. Smith in a discussion on the etiology of primary bronchiectasis expressed the opinion that the Spirocheta pallida could be responsible for sacculated, cylindric or fusiform dilatations and that these were produced in the same manner as aneurysms were produced by Spirocheta pallida. It is his opinion that the bronchi present the sacculated, cylindric or fusiform dilatations characteristic of primary bronchiectasis only after the elastic tissue fibers have been weakened and ruptured by the action of an anaerobic group of organisms which include among its members Treponema microdentium and Treponema macrodentium. It must be appreciated from the onset that all Smith's remarks refer only to the above-mentioned association and do not attempt to explain other forms of so-called primary bronchiectasis in which the spirochete does not play a part. Smith was able experimentally to produce lesions similar to bronchiectasis in man. These lesions were produced in rabbits.

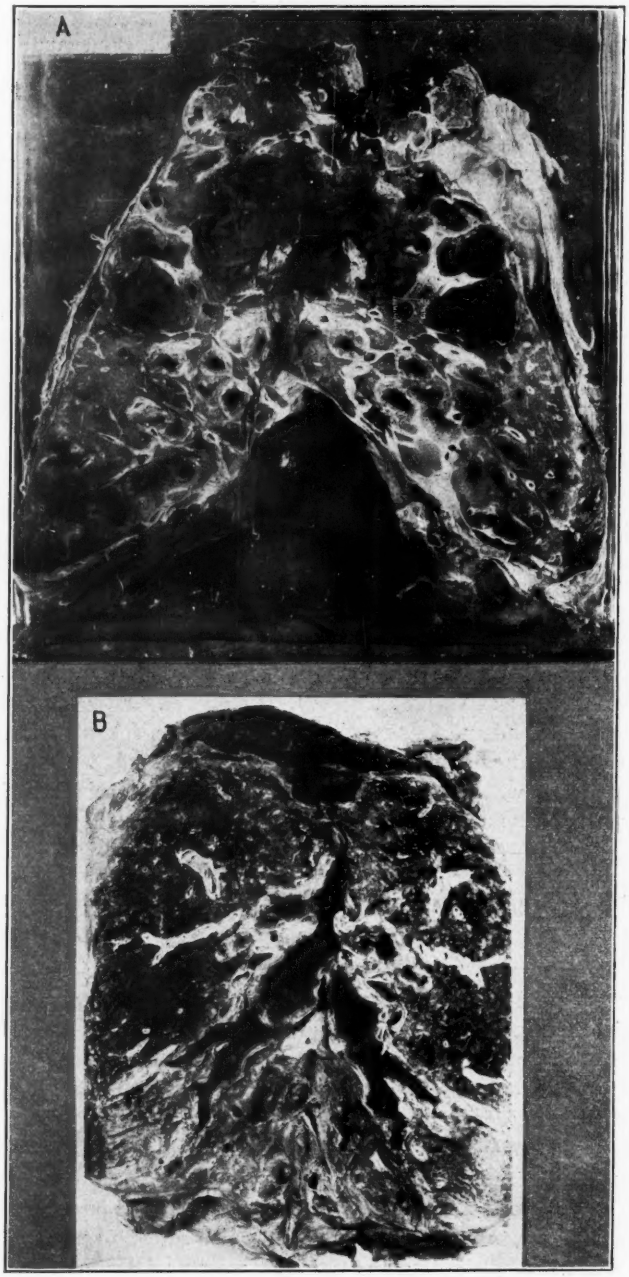


Fig. 10.—*A*, Saccular bronchiectasis of the atrophic type in the right upper lobe, and sclerotic bronchiectasis in the right middle and lower lobes. Note the pneumonic patches throughout the lower lobe. *B*, Sclerotic cylindrical bronchiectasis in a tuberculous lung. Note the extensive basal fibrosis.

With regard to the demonstration of spirochetes and fusiform bacilli in the sputum of patients suffering from bronchiectasis Smith points out that the residual sputum from the depths of the lungs is the most

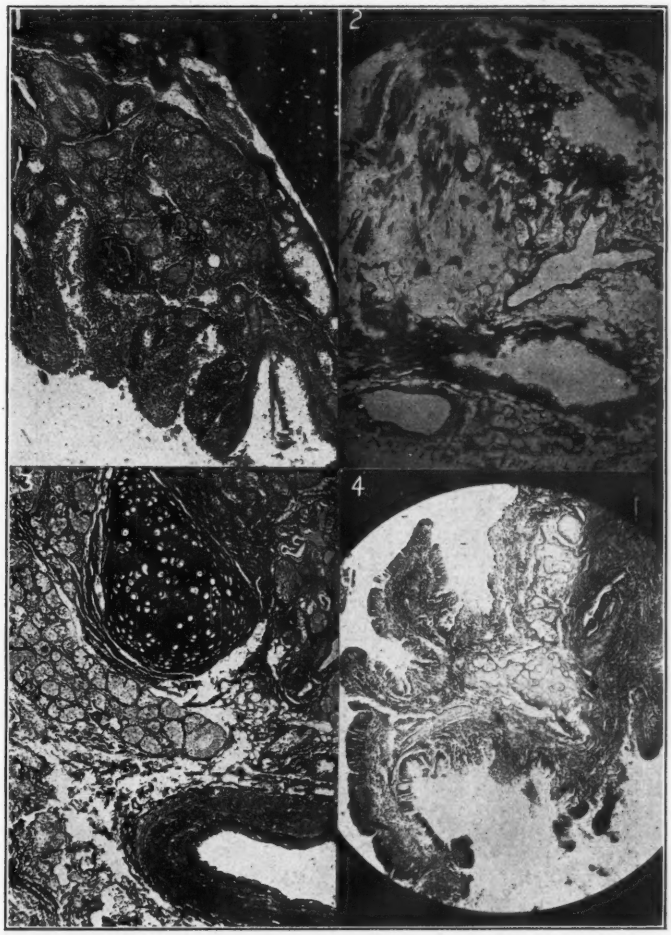


Fig. 11.—*1*, Microphotograph of a section taken from a specimen similar to that in Fig. 10 *A*. Note the marked infiltration of all layers by inflammatory cells. The continuity of the elastic tissue is everywhere interrupted. The cartilage is rather well preserved. *2*, Section taken from one of the sclerotic bronchi in a specimen similar to that illustrated in Fig. 10 *A*. Note the marked destruction of the cartilage and the loss of elastic tissue in this region. The same bronchus may show histologic variations at different levels, for it is exceptional that any given bronchus should show uniform changes. *3*, Microphotograph from a specimen similar to that in Fig. 9 *B*. The microscopic changes referable to the bronchi in this case were few. There is a loss of elastic about the glands and about the well-preserved cartilage. All the above sections have been stained by the Weigert elastic and the Van Giesen connective tissue stains. *4*, Polypoid formation of the epithelium observed in a lung showing atrophic cylindrical bronchiectasis with inflammatory changes. Such a finding is not uncommon and probably accounts for the bleeding in certain cases. In such cases the blood vessels which accompany the bronchi are often found much thickened and their lumens narrowed.

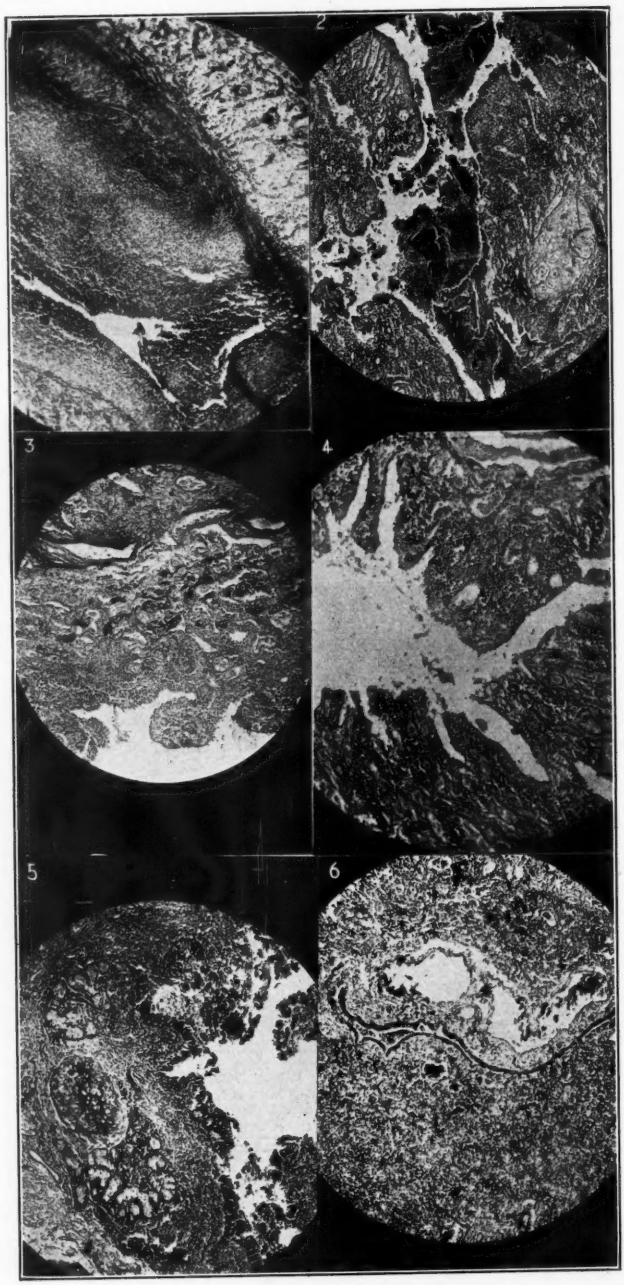


Fig. 12.

desirable, so that he employs the last material raised following postural drainage. In his latest work Smith concluded the following:

"The essential lesion in primary bronchiectasis is a focal necrosis of the elastic tissue in the bronchial wall due to infection with the fusospirochetal group of anaerobic organisms. This fusospirochetal group, comprising T. macrodentium, T. microdentium, S. Vincenti, S. buccalis, fusiform bacilli, vibrios and cocci, are the same organisms that are found in pyorrhea alveolaris, Vincent's angina, pulmonary abscess and pulmonary gangrene."

Smith observed these microorganisms in the sputum of forty-nine of sixty patients proved to have bronchiectasis by the iodized oil test. He found spirochetes and fusiform bacilli in sections of bronchial dilatations after staining by Levaditi's method in eight of twelve cases which came to necropsy and fusiform bacilli were demonstrated in pathologic sections from four of an additional six cases of bronchiectasis after staining by Goodpasture's method.

Employing fusospirochetal material from varied sources (pyorrhea alveolaris, fusospirochetal bronchitis and pulmonary abscess), Smith was able to produce pulmonary abscesses, gangrene and bronchiectasis in rabbits.

"Control inoculations with pure cultures of (1) staphylococcus aureus, (2) aerobic hemolytic streptococci, (3) anaerobic hemolytic streptococci, (4) green producing streptococci, (5) anaerobic streptothrix, (6) Friedlander's bacilli and (7) influenza bacilli failed to produce damage to the bronchi."

Whether or not the microorganisms referred to are secondary invaders is, of course, of import, but as Smith points out their frequent presence cannot be dismissed. And certain it is that some patients with bronchiectasis whose sputum contains spirochetes and fusiform bacilli responded remarkably following the exhibition of neosalvarsan. The response in bronchiectasis is usually in the nature of a reduction in the amount of fetor. The results are more striking when there is profuse expectoration and the tendency toward retention is not great. Unfortunately the amount of expectoration is not influenced

Fig. 12.—*1*, Section taken from a lung showing cylindrical bronchiectasis which was due to bronchial stones. The above microphotograph shows pus in the lumen of a dilated bronchus beyond a bronchial stone. *2*, Microphotograph from a section taken from a specimen showing advanced sclerotic bronchiectasis. The lumen of the bronchus is filled with pus. The elastic tissue system is everywhere interrupted. Infiltration by inflammatory cells is marked throughout. The muscular elements are somewhat hypertrophic. *3*, This section was obtained from one of the cylindrical dilatations of the lung illustrated in Fig. 8 *A*. Note the break in continuity of the elastic tissue (black staining elements). *4*, This section was obtained from a specimen showing a rather advanced saccular bronchiectasis. All the layers of the bronchi were involved in this case, particularly the elastic tissue. *5* and *6*, Microphotograph from a moderately advanced case of sclerotic bronchiectasis which was associated with much fibrosis and chronic pneumonia. The histologic appearance of the bronchi in many respects resembles that noted in Fig. 2. In *6* one observes an almost completely collapsed bronchus, the gross picture in many respects resembling that observed in a case of canalicular fibroadenoma of the breast.

by neosalvarsan so much as one would wish, for there are many cases in which this drug fails to bring relief.

Jackson has attracted attention to the bronchoscopic aspect of ulcerative bronchitis due to Vincent's organisms. He states that the bronchoscopic appearance in this disease differs from that of diphtheria and pseudomembranous bronchitis in that the ulcerative element predominates in the Vincent's infection. The granulations which are to be noted after foreign body sojourn, he stated, are absent. There may be a mild degree of bronchiectasis in the above-mentioned type of case.

### GAS WARFARE AND BRONCHIECTASIS

Conclusions concerning the effect of "gas warfare" on bronchi differ. Barbour and Williams observed dilatation of the bronchi of varying degree in dogs dying acutely of the effects of chlorine gas. The dilatations of the bronchi might have been due (*Medical Aspects of Gas Warfare*, Vol. 14, p. 498) to mechanical obstruction of the bronchi by sloughs of necrotic membrane and exudate. "In the delayed deaths, and more particularly in dogs recovering from severe gassing and killed some time afterward, bronchial dilatation, or rather true bronchiectasis, constituted a very conspicuous feature of the lungs. In these cases it seemed quite clear that the chronic infection with weakening of the bronchial wall, aided by obstruction resulting from the organizing bronchiolitis, was the chief etiologic factor."

### FUNGUS DISEASE AND BRONCHIECTASIS

Fungus disease of the lung is occasionally associated with bronchiectasis.

Pulmonary sporotrichosis has been reported by Forbus, Singer and others. According to Forbus, pulmonary infections by this fungus are not common. Certain it is that bronchiectasis due to pulmonary sporotrichosis is rare. Singer and Ballon have referred to streptothricosis and bronchiectasis. Blastomycosis and actinomycosis are occasionally responsible for dilatation of the bronchi.

### PERIBRONCHIAL FACTORS

Included in this group are enlarged mediastinal lymph glands, mediastinal adhesions, mediastinitis and intrathoracic tumors and aneurysms. We refer at this point only to mediastinal pleurisy and its consequences.

Mediastinal pleurisy which was first described by Laennec, Andral and Cruveilhier is frequently associated with bronchiectasis and usually results from paravertebral bronchiectasis. Mediastinal pleurisy is undoubtedly due to a primary atelectasis in some instances. The

frequent occurrence of mediastinal pleurisy and bronchiectasis has been demonstrated both clinically and radiologically by Rist, Jacob, Troeme and Nobécourt.

Any evidence of mediastinal thickening, particularly in a child, is very suggestive of paravertebral bronchiectasis; only a negative lipiodol injection permits one to exclude its presence in such cases. Strangely enough, mediastinal pleurisy in infants and young children is usually nontuberculous. The same cannot be said for this condition in adults. In them mediastinal fibrous thickening with resultant mediastinal displacement is usually due to a tuberculous process.

### PULMONARY FACTORS

The pulmonary factors include the various types of pneumonia, influenza, "pulmonary fibrosis," abscess of the lung, tuberculosis, anthracosis, etc. Some of the above-mentioned conditions could, of course, be considered in other groups but it is felt that the above classification is simple and serves most purposes.

### PNEUMONIA, INFLUENZA AND BRONCHIECTASIS

The rôle of pneumonia and of influenza in the production of bronchial dilatations has been referred to repeatedly throughout the text up to this point. That many cases of bronchiectasis now observed are undoubtedly the aftermath of the influenza epidemic of 1918 has been appreciated. Still the occurrence of bronchiectasis following influenza is perhaps not so frequent as many would lead us to believe. The frequency of this association should not be exaggerated, for there are many causes of bronchiectasis and not every attack of influenza is followed by it. It is, of course, admitted that influenza may be followed by bronchiectasis. It is well to remember, however, that the influenza bacillus may also appear as a saprophyte in the sputum of patients suffering from bronchiectasis. It is also worthy of note that Lucksch suggests that there may be visible and filtrable forms of the influenza bacillus. Perhaps such forms may be responsible for the production of the dilatations. The dilatations which are due to influenza are usually of the smaller bronchi. Opie, Freeman, Blake, Small and Rivers found influenza bacilli in the terminal bronchi of patients who died from postinfluenzal pneumonia and bronchiectasis (1918). Experimental lesions were produced in monkeys by Blake and Cecil but Smith states that it has never been conclusively proved that the influenza bacillus can destroy the elastic tissue of the larger bronchi. Some would attribute these dilatations to a neuromuscular lesion, the effect of some toxin and peribronchial cell infiltration which results in atony of the bronchi.

It is extremely important that one differentiate between uncomplicated influenza and influenzal pneumonia. The lesion in the former is usually bilateral and regularly involves the trachea in particular and also the bronchial tree. Patches of emphysema and purulent bronchiolitis and bronchitis are frequent findings. Such cases seldom go any further since there is little consequent fibrosis. The picture is altogether different in influenzal pneumonia. Secondary organisms (streptococcus) are frequently present in such cases. There is much necrosis, and lymphatic and interstitial suppuration. As might be expected, bronchiectasis with or without multiple abscess formation is a common consequence. What has been said about influenzal pneumonia also holds true for certain cases of unresolved pneumonia (Fig. 13). We shall refer to these types again when considering the fate of the untreated case of bronchiectasis.

### PULMONARY FIBROSIS

Tuberculosis is a common cause of pulmonary fibrosis in the adult but in children most cases of pulmonary fibrosis are consequent to the bronchopneumonia of measles, grippe and whooping cough. But even the so-called croupous pneumonia may be followed by a similar process, for the connective tissue which develops following such an inflammation has a tendency to shrink and contract. Brauer has repeatedly pointed out that the pneumonias which follow the exanthemata and other infectious diseases frequently result in small peripheral abscesses which later form the so-called caverniculae form of bronchiectasis.

That many fibrotic lungs in children are due to aspirated foreign bodies is well known. Tegteier has reported a dextroversio cordis which developed as a result of a foreign body's being aspirated during early childhood. We have also observed such cases associated with bronchiectasis.

The mechanics which are responsible for displacement of the mediastinum and its contents are somewhat as follows: The loss of elastic tissue causes a reduction in the volume of the lung. In this way an excessive unbalanced pull will act on the larynx, trachea, heart, mediastinum and diaphragm. The shrinking of the lung is followed by a pulling over of the mediastinum and its contents to the affected side. If there is, in addition, pleural involvement, then the shrinking will be more marked. The position of the diaphragm, the presence or absence of adhesions will also play some rôle as to the position which the heart is eventually to occupy, since elevations of the right diaphragm usually displace the heart to the opposite side while elevations of the left diaphragm may be associated with displacements and rotations of the heart. The opposite lung develops the so-called vicarious emphysema.

The final picture resembles that of cirrhosis pulmonum and in an adult would immediately be suspected as being chronic fibroid phthisis. In a child it is not only strongly suggestive of, but practically always means, nontuberculous bronchiectasis and chronic mediastinitis. Since the above is not of infrequent occurrence, it justly raises the question as to whether bronchiectasis should be prophylactically treated during childhood. In other words, should children who have persistence of cough and expectoration following some infectious disease or pneumonia be treated early? Should they receive pneumothorax treatment? Might one so avoid the terrible consequences of fibrosis, infection and displacement of the heart and other mediastinal contents? It is certainly wise to subject children who have persistent unexplained cough and expectoration to bronchoscopic examination early to exclude the possibility of foreign body and to avoid the consequences of bronchostenosis. Further reference to pulmonary fibrosis and bronchiectasis will be made when discussing the prognosis of such untreated patients.

Fig. 13.—Chronic pneumonia and bronchiectasis. The above specimen illustrates the picture of a relatively chronic pneumonia. The lower half of the lung is rather well preserved. If the lesion in the upper half would progress, then it would form one of the severer forms of bronchiectasis, a type which is always associated with repeated attacks of pneumonitis. The overlying pleura is considerably thickened.

### ABSCESS OF THE LUNG AND BRONCHIECTASIS

Abscess of the lung and bronchiectasis are frequently associated. And it is worthy of note that M. Briquet years ago made the interesting observation that in certain cases of pulmonary fetor the extremities of bronchial tubes became dilated and gangrenous independently of any other portion of lung tissue. He cites several cases. From his description one cannot exclude the possibility of an infected cyst which communicated with a bronchus and was lined by bronchial mucous membrane. Dietrich of Erlangen also connected certain putrid infections with dilatations of bronchi.

The following classification of abscess of the lung based largely on the roentgen-ray findings, and previously published, notes some associations of abscess of the lung and bronchiectasis (Ballon).

#### CLASSIFICATION OF ABSCESS OF THE LUNG

I. Solitary
   (a) With fluid level. This type of abscess is demonstrable by ordinary roentgen ray.
   (b) Large bronchiectatic abscess, acute or chronic. This type of abscess may be aspirated and may be injected with lipiodol. It is often bilateral and is at times associated with some other phase of bronchiectasis.
   (c) Associated with bronchiectasis, chronic. This is of the narrow neck type in which expectoration is but overflow. It shows no fluid level and on injection the abscess area casts no shadow of the lipiodol.

II. Multiple
   (a) Multiple lung abscess, chronic, which may be associated with bronchiectasis.

III. Secondary
   (a) Those due to tumor and foreign body.

IV. Tuberculous
   This type may be associated with bronchiectasis, acute or chronic. Metastatic lung abscess and cyst degeneration are not considered in this classification.

A solitary lung abscess may thus be associated with bronchiectasis. When the former is treated early, then the latter usually becomes healed. If the abscess does not heal and if it is neglected for many months, then the associated bronchiectasis may become very obstinate. In this connection it is well to point out that the occurrence of a febrile disease with cough and fetid expectoration following immediately upon an operation in a patient who had no previous respiratory infection is not necessarily sufficient to make a diagnosis of abscess of the lung, for the condition may be bronchiectasis.

It is not enough to assume the presence of an associated bronchiectasis in those patients in whom physical and roentgen-ray signs suggest more than abscess. Abscess of the lung is in many instances

complicated by demonstrable bronchiectasis. It is always important to inject the sinus of a patient operated upon for abscess of the lung. Many abscess cavities are of the narrow necked type, the communicating bronchus being edematous and filled with granulations. Expectoration is but overflow. They do not show a fluid level and are not amenable to bronchoscopic aspiration. Hence, the lung area containing the cavity remains as a blank and fails to be injected with lipiodol; the dilatations, therefore, are demonstrable only after operation. Cases which are illustrative of abscess of the lung and bronchiectasis will be presented when discussing treatment. Large bronchiectatic abscesses are always associated with smaller abscesses. The latter need not be bronchiectatic in nature, by that is meant that they need not be lined by bronchial mucous membrane but may be peribronchial communicating with bronchi or actual excavations in the lung. In the bronchopneumonias of measles, pertussis, etc., the supporting network of the lung may be damaged and abscess formation and bronchiectasis may be the result. It is therefore undoubtedly true that bronchiectasis may be a secondary manifestation of multiple lung abscesses or pneumonitis in such cases. The reverse also holds true. Whether or not the bronchiectasis is primary or secondary, local or diffuse, is early of considerable import from the standpoint of prognosis and treatment and will be referred to again when discussing treatment.

### PULMONARY TUBERCULOSIS AND BRONCHIECTASIS

The same factors which ordinarily operate in the production of bronchiectasis do so in pulmonary tuberculosis. The "mixed infection" in pulmonary tuberculosis involves not only the mucous membrane but also the various coats of the bronchial tree. It is the mixed infection which causes the ulceration. The tendency to heal with its resultant fibrosis and shrinking must of necessity produce dilatations of the bronchi.

Tuberculous peribronchial glands may either break into a bronchus or so narrow it as to produce dilatations beyond the point of narrowing.

Following extrapleural thoracoplasty, the bronchial tree may become a large tube with dilatations at the base. Many of these dilatations are really the associated bronchiectasis which is so frequent a finding in a suitable case for such a procedure. Compression may further accentuate them. The degree of associated bronchiectasis and atelectasis is, therefore, of considerable import.

McCrae and Funk have reported examples of apical bronchiectasis associated with tuberculosis. Chronic bronchiectasis terminating in phthisis florida has been reported by Bray and others. The old rule

that tuberculosis is apical and bronchiectasis basal still holds true in most instances, and isolated apical bronchiectasis is usually tuberculous. It must always be remembered that bronchiectasis is but a state of the bronchi not a disease in the strict sense of the word. Hence, a lipiodol injection which demonstrates the presence of bronchial dilatations does not always permit one to exclude the possibility of pulmonary tuberculosis.

### PNEUMOCONIOSIS AND BRONCHIECTASIS

The clinical picture of pneumoconiosis may be that of bronchiectasis. Anthracosis, chalicosis and silicosis will all be considered under this heading. In a recent review based on the study of 208 examinations among rock drillers, blasters and excavators in New York City, silicosis was found in 57 per cent of the men examined. Large areas of massive pulmonary fibrosis were not infrequently observed but no mention is made of bronchiectasis.

As Ickert and others have shown, dust of various kinds inhaled into the lung tends to be carried to the pleura and lymph glands. In the pleura such scattered accumulations may roentgenologically suggest miliary tuberculosis. The resultant obstruction and blockade of the lymphatics usually lead to fibrosis. In addition pseudotubercle formation may take place in the lung and fibrosis as well.

It is worthy of note that bronchial stones have never been observed in a case of pure pneumoconiosis, that is, without tuberculosis. When present, there is active pulmonary tuberculosis. The theories as to the action of these various dusts include "allergic," infectious (Sternberg), etc. Many stress the necessity of an associated bacterial action. Concerning the fibrosis which interests us most, it can be said that it requires years to develop. Schudde considers the fibrous tissue in such cases to be similar to keloid of the skin. He feels that a special predisposition is necessary. Anthracotic glands may compress a bronchus sufficiently to produce dilatations early.

Areas of colloquation necrosis in the lung may produce dilatations of the terminal bronchi beyond them. Such examples have been observed.

Periadenitis and fibrosis are not infrequent following the inhalation of asbestos dust. Granite dust is said to produce an increase in the size of the lymphoid tissue in the upper respiratory tract. Although tobacco dust may cause bronchitis, it does not produce positive roentgenologic evidence of fibrosis.

Jarvis states that in the worker in a dusty trade, adaptation of the respiratory tract lost and regained many times often acts as a suit-

able preparation for developing a serious respiratory condition. In this connection mouth breathing is not as injurious as nasal respiration.

"Of twenty-one potters whom I have had under observation, the evidence of dilatation of the bronchi was present in ten." (Norris and Landis.) These authors have also stated that pneumoconiosis rarely, if ever, develops as a result of exposure to organic dust. They feel that this form of dust is incapable of producing a mechanical injury, and, further, that it is doubtful if any of it ever reaches the finer bronchi and lung cells. On the other hand, they are of the opinion that one of the most frequent causes of bronchiectasis is the prolonged exposure to inorganic dust.

### PLEURAL FACTORS

There does not seem to be much evidence to support the idea that a pleural effusion, adhesions or pneumothorax can of themselves be responsible for the production of bronchial dilatations. The cause of bronchiectasis associated with a pleural effusion or discovered following the treatment of the latter is always to be sought in the lung and not in the pleural cavity. A bronchopleural fistula which persists for any appreciable length of time may result in the production of bronchial dilatations in the adjacent lung tissue.

### THORACIC CAGE

The relationship of the thoracic cage to the production of bronchiectasis was referred to when considering bronchiectasis during childhood.

*(To be continued.)*

# BRONCHIAL STENOSIS*

*(To be published in three installments)*

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## I

CONSTRICTIONS of the hollow viscera have a well-defined pathology and symptomatology in their train: for example, dilatation of the biliary channels and gall bladder follow stricture of the common duct, hydronephrotic changes follow ureteral stricture, changes in the heart and blood vessels follow stenosis of the various cardiac orifices. Bronchial stenosis, however, has not had the attention its importance in the explanation of frequent yet obscure clinical phenomena and pathologic pictures merits.

Not that bronchial stenosis is unknown. Obstruction due to foreign bodies and its sequels has been accurately and completely described, notably by Chevalier Jackson,[1] Manges and their coworkers of the Philadelphia school: thanks largely to them its clinical, bronchoscopic and radiographic manifestations are well known, both in the acute and chronic stages. Massive pulmonary collapse, referred by many authors to massive acute bronchial obstruction, has been the subject of intensive study in the last few years.

It is not of these that this study will endeavor to treat, but of chronic stenosis, partial and complete, steadily or intermittently progressive, its causes and effects.

This kind of stenosis has also had its investigators, as indeed it is impossible that so common a disorder should not have been studied and described. Oppikofer[2] of Basle made wax casts of 240 specimens of the larynx, trachea and bifurcation; his tabulated measurements furnish a valuable anatomical basis for pathologic studies. The late Carl Hart[3] treated the anatomical aspects of stenosis so exhaustively in Henke-Lubarsch's large *Handbook of Pathological Anatomy* that little need be added. E. Mayer[4] completed Hart's work after his death. A number of clinicians, beginning with Traube[5] and Lichtheim,[6] and, following them, Gerhardt,[7] Lewis Conner,[8] Fraenkel[9] and Pitt,[10] have dealt with the symptomatology and have studied the respiratory changes of stenosis clinically and experimentally, and more recently Hoover,[11] Nissen[12] and others have added valuable experimental researches. It seems strange that from all these finished fragments the

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various clinical pictures should not have been set, but Sauerbruch[13] in his very thorough *Surgery of the Thoracic Organs* mentions only the pathophysiologic and radiologic aspects of stenosis; Lilienthal[14] gives it no mention at all. Hoffmann[15] in Nothnagel's *System of Medicine* devotes a few scant pages, mainly to a citation of the various causes of stenosis; Lord's[16] textbook is still more brief, and McPhedran[17] in McCrae-Osler's *Modern Medicine,* while devoting a three-page chapter to the subject, considers only high stenosis and stenosing bronchiolitis and leaves the intervening territory untouched. Chevalier Jackson has given chronic stenosis of the trachea and bronchi more consideration; references are to be found in his textbook[1] and other writings.* Most of these authors occupy themselves only with tracheobronchial stenosis; unilateral and unilobar stenosis they do not consider. The bronchoscope and the x-ray with lipiodol have led to an accuracy of diagnosis impossible before their discovery; recent experimental work has added to this mass of material; bronchoscopists, radiologists and experimenters have forced a great number of isolated facts upon the clinician. So that clinical pictures which do exist in fact might be built up hypothetically from evidence already at hand. It is to these clinical pictures which I fail to find set forth that I should like to draw attention, feeling certain that many of us are unaware of the frequency of stenotic types of pulmonary disease.

## CAUSES AND LOCALIZATION OF STENOSIS

*Congenital Stenosis.*—Bronchial stenosis may be congenital or acquired, and either form may be due to pressure from without or narrowness within. Sauerbruch[19] and his assistant Lotzin have called attention to congenital anomalies as a cause of bronchiectasis. Many they consider to be due to an unusually high or unusually persistent duct of Cuvier, a connection between the peripheral venous sinuses and the heart over which the left lower bronchus rides. They consider that the anatomical relation between the duct and the lower left bronchus accounts for the predominance of bronchiectasis in the left lower lobe and they have described distinctions in the microscopic anatomy of acquired and congenital dilatation. The ridge formed by the duct exerts pressure on the left lung bud or its stalk. If pressure occurs early in the course of fetal development the whole left lung may be deformed; later the larger bronchi and their endings; later still the changes may affect the left lower lobe only, which in any event bears the brunt of the pressure. Heller[20] and Orth[21] look upon dilatation of the bronchi as secondary and attribute it to traction of the rapidly growing chest wall and diaphragm which, finding little expansile alveolar tissue in the maldeveloped underlying lung, exert their tractile force on the bronchi. Thus an early congenital anomaly

*Since this paper was written, Haslinger[18] has published an exhaustive treatise on organic stenosis of the trachea and bronchi. His paper contains a full bibliography.

may involve the whole lung with obliteration of the entire bronchial tree and conversion of the lung into a huge uni- or multilocular cyst, which is the form termed by Grawitz[22] bronchiectasis universalis. A later anomaly may involve merely a few bronchi of the second or third order, the result being small intrapulmonary cysts, designated by Grawitz as bronchiectasis teleangiectatica. The anomaly may involve the bronchus to a rudimentary lobe with cystic degeneration of this rudiment, which usually lies at or near the mediastinum. Such a cyst may in turn lie against the wall of a large bronchus and compress its lumen.

Rach[23] relates the case of an infant with a stridor as of a tubercular mediastinal lymphadenitis. X-rays revealed nothing abnormal. The

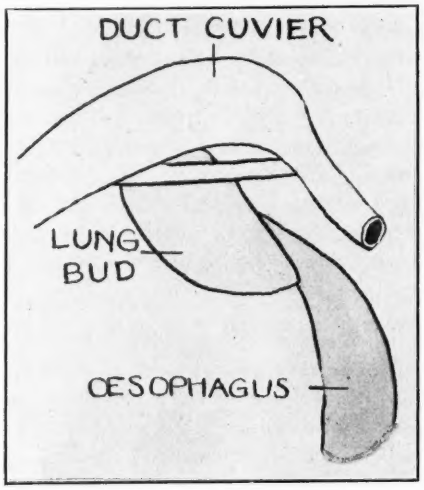



Fig. 1.—Duct of Cuvier.

child died of pneumonia. Autopsy disclosed a cyst the size of a walnut lying at the bifurcation and compressing the right main bronchus. It contained mucocolloidal material and was lined with ciliated epithelium; a communication with the trachea or bronchi was not to be found. The secondarily obstructed right lung is not described.

Variations, like congenital variations of the lobes of the lung and the bronchial distribution, are almost innumerable. The various congenital anomalies and the question of their origin are too intricate to be discussed at length; not only may outgrowth of the pulmonary bud from the primitive digestive tube be disturbed, but particles of lung tissue (lung anlage) may become separated and lie detached from their primitive stalk, giving rise to mediastinal mucous cysts lined with tracheal epithelium. (Cf. Schneider in Schwalbe's *Handbuch der Misbildungen.*) These cystic degenerations, atelectases and bronchi-

ectases, are to be considered as accompaniments or results of various congenital stenosing deformities of the bronchial tree. They are probably not at all infrequent.

CASE 1.—A student of twenty was referred by Dr. James Ward in 1927. In 1917, at the age of nine, he had had fever and cough. What was thought to be

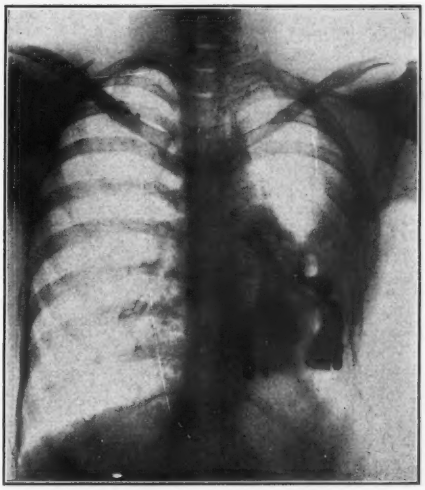


Fig. 2.—Case 1. Congenital cystic lung. Lipiodol injection.

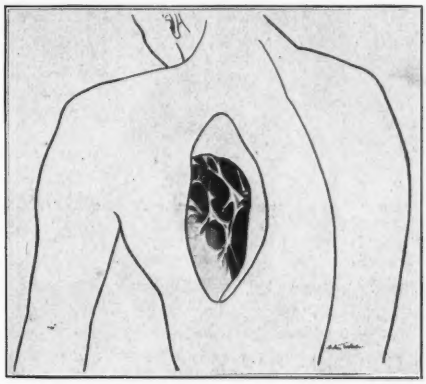


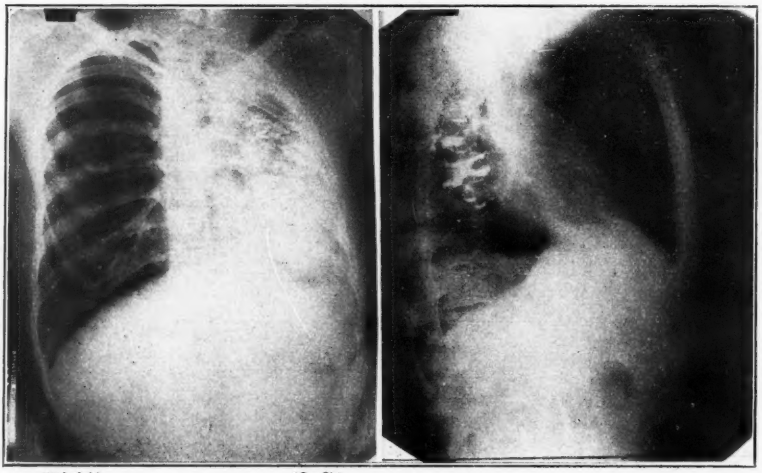
Fig. 3.—Case 1. Congenital cystic lung, after opening. (Drawn from photograph.)

an empyema was opened and drained in 1920. In 1927 I found a flat retracted left chest which did not move on respiration. There was a drainage tube in the course of the tenth rib which communicated with a bronchus. The heart was displaced to the left. Injection of lipiodol demonstrated a constriction just below the origin of the left major bronchus, past which the oil dripped slowly to form a bilocular puddle at the summit of the diaphragm. The puddle had a clearly demonstrable connection with two large bronchi.

At operation the lower lobe was found to be represented by a conglomeration of large cysts with a red velvety lining; connected at one point with a bronchial opening. The upper lobe was converted in a huge cystic cavity which was separated from the lower cystic lobe by a tough membrane about ¼ inch thick, containing a narrow layer of spongy lung tissue in its center. The upper cavity had a pearly white, glistening lining, pierced by a number of thin slits, representing flattened bronchial mouths.

The cysts of the lower lobe were opened and drained and finally the whole lobe resected. The upper air-containing cyst was left untreated. The lad made a good recovery.

CASE 2.—M. P., an Italian of twenty years, entered the San Francisco Hospital, April 15, 1929, complaining of a dry cough of one week's duration. Four days previously he had brought up a tablespoonful of bright red blood. The left chest was thin and retracted. The apex was dull. The breath sounds were diminished and there were many râles on the whole left chest. The man was bronchoscoped



(Right) Fig. 4. (Left) Fig. 5.

Fig. 4.—Case 2. Congenital cystic lung; cysts filled with lipiodol showing marked retraction of mediastinum to left and contraction of left chest.

Fig. 5.—Case 2. Same, lateral view.

by Dr. Tillotson. The carina occupied an oblique position. The left main bronchial orifice was reddened and too small to admit the tip of a 7 mm. bronchoscope. A small anomalous orifice was noticed opening into the left lateral wall of the right major bronchus which, upon lipiodol injection, was seen to communicate with the left major bronchus.

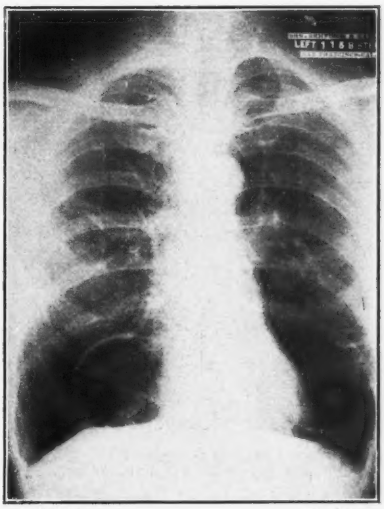
The patient had no fever. No tubercle bacilli were found. He was discharged without further treatment.

CASE 3.—A retired saloon keeper of sixty consulted Dr. Arthur Sonnenberg for rectal bleeding. He had had occasional attacks of cough and fever, thought to be grippe, to which he had paid no attention. Examining the chest, before proceeding with gastrointestinal roentgenography, Dr. Rehfisch was struck with an annular shadow and reported a delicate linear shadow marking off a segment of lung anteriorly which suggested a cystic middle lobe. Approximately 20 c.c. of lipiodol

were injected. The injected bronchi completely surrounded the thin-walled sac in the right lower lobe and further brought out a small peripheral cyst at the diaphragmatic angle. It was impossible, however, to inject the sac itself, the bronchial opening being too fine to admit the oil. The chest was not treated.

Obstruction must be incomplete if it is to produce a large air-containing cyst, although the connecting channel may be very narrow. Total obstruction leads to atelectasis or a mucous cyst.

Acquired strictures may affect the respiratory channels from the glottis down to the bronchioles with effects varying according to the level and degree of obstruction. Laryngotracheal strictures do not concern us here.



(Right) (Left)

Fig. 6.—Case 3. Congenital cyst, right base.

*Tracheobronchial stenosis and stenosis of the larger bronchi* occur both from outward pressure and intrinsic processes. v. Schrötter[24] divides their causes into: extramural (compression), mural (tumors, inflammatory strictures, cicatrices), and intramural (foreign bodies).

*Compression Stenosis:* Among the causes of outward pressure are enlargements of the mediastinal viscera of various kinds; deep-lying goiters, both innocent and malignant, mediastinal tumors, sarcoma of the lymph glands, carcinomas of the esophagus and of thymus remnants, osteomas and chondromas of the vertebral column and of the sternum and metastatic tumors; cysts, such as the cysts mentioned above, dermoids and echinococcus; dilatations of adjacent mediastinal viscera, of the heart, the great vessels, possibly also of the esophagus; inflammatory processes, lymphadenitis, mediastinitis, mediastinal pleurisy and suppuration of various kinds originating in the mediastinum or breaking

through into it: and finally, sclerosing processes and scars, results of the foregoing inflammations.

A goitrous enlargement may lie entirely within the chest and not be discernible above the sternal notch. A characteristic mediastinal dullness and corresponding shadow in the x-ray may betray its presence. Goitrous compression occurs comparatively often in the young; cancerous goiters make the most marked stenosis. Stenosis is notoriously without relation to the size of the goiter, but depends on the firmness of its attachment to the tracheal wall and its position, retrotracheal thyroids being especially dangerous. Whether goiter causes a characteristic tracheomalacia is questionable, but certainly softer youthful tracheas are more prone to be distorted than senile, less yielding ones.

Fig. 7.—Thymoma showing large masses compressing veins, but leaving trachea intact. Trachea injected with barium gelatin.

Tuberculous mediastinal pleurisy, interlobar empyema and various suppurations of adjacent organs tending to break through into the mediastinum may make similar stenosing infiltrates.

Whether pressure from persistent thymus can compress the trachea and cause sudden death has furnished a theme for much heated argumentation, the substance of which is that it may, but rarely does. (Klose.[25]) Hart[3] denies the possibility in adults: he concedes it in infants who are unable to hold their heads up, and in young children during narcosis. Compression is more likely to occur at the upper thoracic aperture when the neck is hyperextended; screaming and struggling may engorge and further enlarge a hyperplastic thymus.

Mediastinal lymphosarcomas, thymomas and Hodgkin's tumors usually compress the trachea and bronchi fairly late in their course. The soft vascular masses compress the veins first; cyanosis and congestion of the upper part of the body are primarily due to venous engorgement rather than blocking of the air passages. Carcinomas in the

mediastinum, esophageal carcinoma, and metastatic tumors are smaller and firmer; they may compress the main bronchi and trachea before seriously encroaching on the great veins. Of these esophageal carcinoma is the most common, yet it rarely causes bronchial obstruction, although it often breaks through into the air passages and the aorta. Every radiographer has films showing aspiration of a barium meal through a tracheo-esophageal fistula, but as a rule ulceration and destruction precede and prevent constriction. When stenosis does occur, it is as likely to be due to pressure of metastatic glands as to the esophageal tumor itself. Other rarer tumors, osteomas of the spine, ganglion-neuromas of the sympathetic chain, etc., are to be considered.

Pressure from congenital pulmonary cysts has already been mentioned; paratracheal diverticula and cysts are of kindred origin; dermoids and echinococcus cysts may also compress the air passages.

Common among causes of extrabronchial pressure are dilatations of adjacent mediastinal viscera; the heart, the aorta, and occasionally the esophagus. The brassy cough and pulmonary congestion accompanying aortic aneurysm are well known (Osler's "aneurysmal phthisis"). Aneurysms of the descending aorta and anonyma and of the lower posterior portion of the arch compress and perforate the trachea and the left main bronchus, those of the ascending portion of the arch, the right main bronchus. Heart disease (mitral stenosis) with dilatation of the left auricle compresses the left main bronchus.

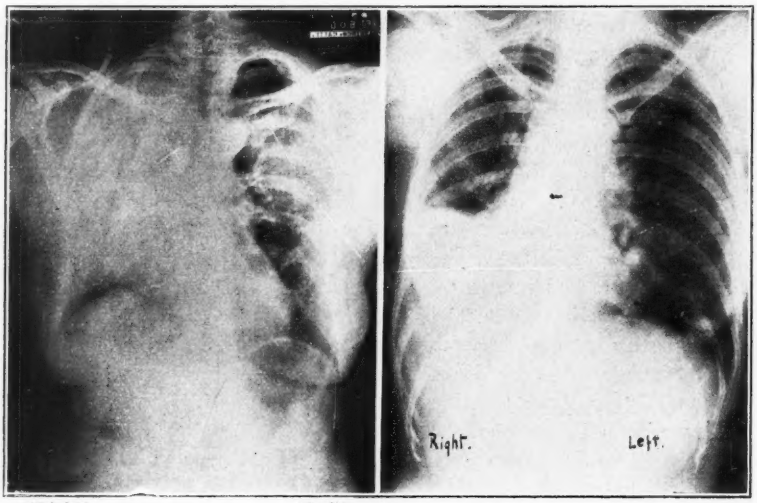
That pulsion diverticula of the esophagus rarely cause stenosis is due mainly to their location. The stenosis accompanying traction diverticula is less a sequel of the diverticulum itself than of the underlying cause, a mediastinal lymphadenitis or paratracheal cyst, as will be seen later.

Extrinsic inflammatory causes are frequent. This is not surprising; the mediastinum is a cesspool where products of all inflammations that beset the chest and some of the abdominal ones gather. Enlargement of the mediastinal glands, both tuberculous and nontuberculous, is very common. In the process of suppuration or fibrosis the glands attach themselves to the bronchi; they may compress them; they may kink or distort them; they may finally, following a preliminary period of constriction, perforate. And not only the glands themselves may perforate, but they may adhere to other structures and draw them with them. Thus the aorta or pulmonary artery may perforate into the trachea or bronchi and so may the esophagus following establishment of a traction diverticulum; or a communication may be established between all three, first between the esophagus and the air passages, and into these in turn the aorta may perforate.

It is doubtful whether all esophagotracheal perforations and all esophageal traction diverticula at the level of the bifurcation need the intervention of an adhesive lymphadenitis. Remnants of congenital

esophagotracheal adhesions, comparable in origin to congenital esophagotracheal fistulas may persist, and abetted in later life by superimposed inflammatory processes may lead to diverticula and even to perforation. Small paratracheal diverticula and cysts of similar congenital origin, which seem to be common, may account for further cases of compression and perforation.

An old fibrous mediastinitis may cause difficulty both in swallowing and breathing without the intervention of lymphadenitic swelling. The obscure, senile, noncancerous disturbances of deglutition (so-called senile esophageal stenosis) may be due to this cause.




(Right) (Left)

Fig. 8. Fig. 9.

Fig. 8.—Case 5. Stenosis, right side. Calcified gland to right of tracheal bifurcation.

Fig. 9.—Case 6. Arrow points to chalky gland (retouched) at right bronchial bifurcation. Marked contraction of right chest, retraction of mediastinum with complete dextrocardia and elevation of right diaphragm. Atelectasis of right middle lobe.

CASE 4.—A marine engineer, first seen in 1927, had had difficulty in swallowing since 1918. In 1922 the passage of all food was so difficult that a gastrostomy was done. The x-ray showed a narrowing at the junction of the middle and lower thirds of the esophagus. When last seen in June, 1929, the man could swallow liquids with some difficulty, but still fed himself through his gastrostomy tube. The fact that eleven years had passed since the onset of his trouble precluded the possibility of cancer.

Compression and distortion of the lower trachea and bronchi from adjacent lymph glands have been repeatedly observed and described. They occur in children from tubercular glands and in the aged from nontubercular sclerotic ones. Spongy hyperplastic glands rarely cause stenosis; compression comes with periadenitic tracheal adhesions, caseation and signs of beginning perforation.

CASE 5.—A seamstress of sixty-seven was seen in January, 1928, with Dr. Felberbaum. She had had attacks of coughing since the age of fourteen; she brought up a thick, ropy, yellow sputum which after severe attacks of coughing was sometimes frothy, but never foul. The attacks would last from three to four months; in the interval she was fairly well. She was always short of breath on exertion. Her fingers had been clubbed as long as she could remember. She had never spat blood until two or three weeks before being seen, when she suddenly had a severe hemorrhage.

The patient had a constant racking cough and brought up large amounts of foul yellow sputum. The right side lagged and was dull. The breath sounds over the right top were fairly loud and amphoric; over the right base they were diminished. There were coarse bubbling râles at the right base. In roentgen films the whole right side was seen to be airless; to the right of the tracheal bifurcation lay the shadow of a chalky lymph gland. On the day upon which she was to have been bronchoscoped she had a violent hemorrhage and died. Autopsy was not obtained.

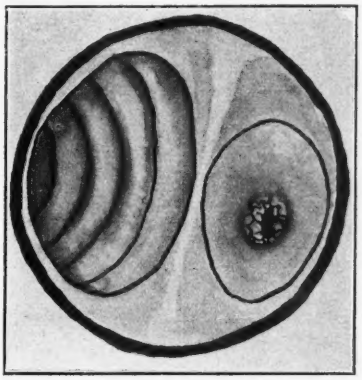


Fig. 10-*A*.

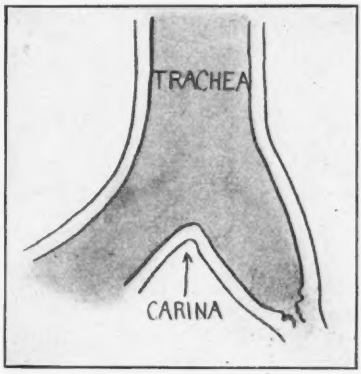



Fig. 10-*B*.

Fig. 10-*A* and *B*.—Case 6. Diagrammatic sketch of ulcerative tubercular stenosis of right main bronchus.

CASE 6.—A stenographer of forty was seen in September, 1928. She had been hoarse, had coughed for three and one-half years, and had a tubercular fibrosis with cavitation of the right apex. In the x-rays chalky tubercular glands were visible at the tracheal bifurcation.

She was hospitalized at the Community Hospital, San Mateo, where Dr. Knorp kindly asked me to see her again on March 27, 1930. She had suffered much from attacks of wheezing and breathlessness. The left side of the chest was hyperresonant, the resonance extending over the midline into the right chest. There were dullness and crepitations over the right hilum. The right anterior intercostal spaces retracted on inspiration. There was a dull area corresponding to the right middle lobe, but this extended so far backward that it sounded as though there might have been fluid in the interlobar fissure. Over this area one heard a distant bronchial whiff. The heart was retracted far to the right.

Bronchoscopy on April 4, 1930, disclosed a normal larynx. The left major bronchus was somewhat flattened on its lateral aspect, otherwise normal. On the right side one looked into a funnel. The bronchial mucosa was red and boggy and the normal rings were not to be seen. About 2 cm. below the bifurcation the funnel narrowed into an annular stricture with a lumen about 4 mm. in caliber. The

edges of this stricture, especially on the left and upper aspect were formed by an ulcer into which chalky white, irregular masses protruded. One could distinctly see the walls widen on inspiration and close on expiration like the mouth of a carp.

In children and young adults tubercular glands not uncommonly ulcerate into the trachea or bronchial bifurcation and are discharged after a stormy struggle with stenotic symptoms.

CASE 7.—A power lineman of 39 had what was called influenza, without cough but with fever, in 1921; he recovered perfectly from this. In 1922 he had a second attack, also without cough. He was sick for two or three weeks with fever, pain in the joints, and shortness of breath and has never been quite well since. In January, 1929, he had a pneumonia and fever to 102-3°. He was so short of breath

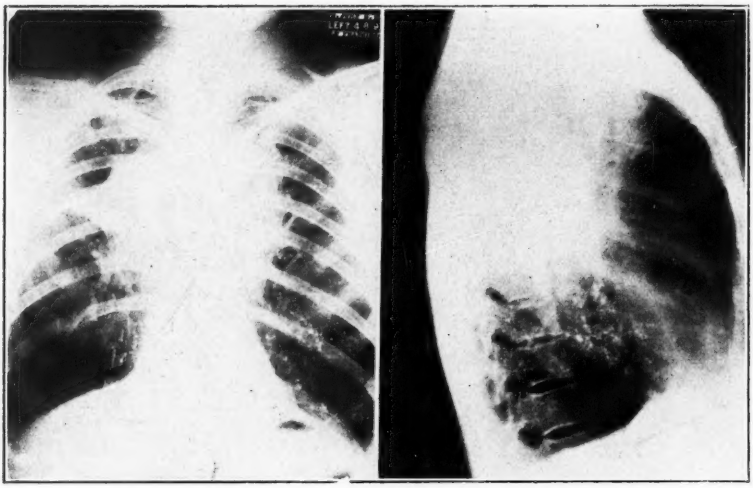


Fig. 11. Fig. 12.

Fig. 11.—Case 7. Large peritracheal mass of lymph nodes with tracheal stenosis.
Fig. 12.—Same as Fig. 11, lateral view.

that his wheezing could be heard all over the house. He had not much cough or sputum. On examination in September, 1929, I found a thin, pale man with clubbed little fingers. He had an inspiratory stridor; there was no dullness and no râles. X-ray showed a large globular mass in the upper posterior mediastinum, pushing the trachea toward the front. Exploratory mediastinotomy revealed a chain of greatly enlarged and adherent peritracheal lymph nodes, the largest twice the size of an English walnut. This gland was removed. Sections showed old scarred tubercles.

There is a large pediatric literature[26] concerning this disease.

Tubercular lymphadenitic abscesses occur at the top of the sternum, occasionally causing compression signs and making a puzzling x-ray shadow; more frequently they lie lower down along the course of the trachea and at the bifurcation.

CASE 8.—May 10, 1926. A Mexican lady of thirty-two had influenza in 1922. She brought up some blood at this time. Up to about fifteen years ago she used to suffer from asthma, but not of late. For the last two or three months she had had pain in her right shoulder and neck. About two months ago she had a tooth pulled and awoke from the anesthetic to find a lump to the left of the sternum. She had a dry cough, brought on especially by pressure on the sternal swelling. The left supraclavicular region was swollen; there was a mediastinal dullness. X-rays revealed a shadow in the upper mediastinum. This was interpreted as an endothelioma of the sternum, but it probably was not, as the patient gave birth to a healthy baby a year later and still remains well.

Lower mediastinal glands breaking down into cold abscesses may perforate and make parasternal sinuses through the fourth and fifth intercostal space. These are not infrequent in middle age. When such

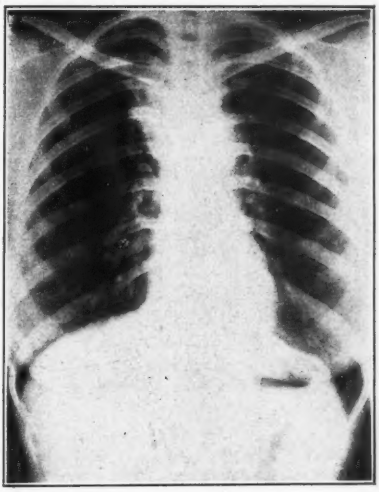


Fig. 13.—Case 8. Upper mediastinal mass with tracheal compression; tubercular lymph nodes.

a sinus is present the diagnosis is as good as made; although suppurating dermoids, cysts, empyemas and also perichondritic suppuration, may make similar perforations and are to be borne in mind.

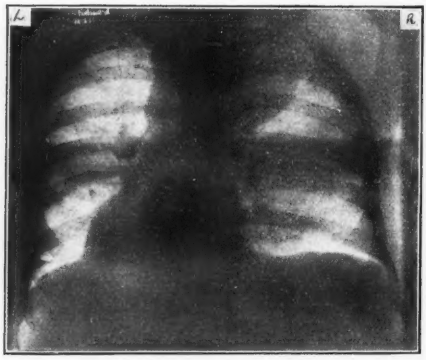
Tubercular mediastinal pleurisy, interlobar empyema and various suppurations of adjacent organs tending to break through the mediastinum may make similar stenosing infiltrates. The following patient had a Pott's abscess discharging into the bronchi:

CASE 9.—A former medical student of forty-two had suffered since 1904 from pulmonary and spinal tuberculosis. In 1921 he had a sense of contraction around the trachea with occasional small quantities of purulent sputum. In 1922 the purulent expectoration increased and a sinus, which had been present since 1921, opened and closed at nine-day intervals. Graphic records show a reciprocal relation between the drainage from the sinus and the bronchial irritation very clearly. When the patient expectorated the sinus drainage was less, and vice versa. Green

soap injected into the sinus was promptly coughed up. Occasionally he could blow air out from the sinus. He still has occasional periods of bronchial irritation and occasional drainage from the fistula; both of these have nearly ceased. He notes that being up favors drainage through the sinus, although drainage may break through the bronchial fistula after many months of quiescence without warning. Sometimes the appearance of purulent sputum is preceded by several days of bronchial irritation and bronchial secretion.

The following staphylococcic vertebral osteomyelitis caused an abscess with great mediastinal thickening, signs of bronchotracheal irritation, compression of the right upper lobe bronchus, and atelectasis of the right upper lobe.

CASE 10.—A Scotchman of forty-four had a perilous staphylococcic bacteremia following a furuncle of the chin. Among numerous osteomyelitic foci he developed



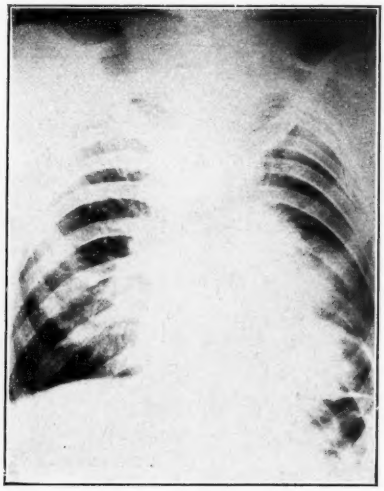


Fig. 14. Fig. 15.

Fig. 14.—Case 10. Staphylococcus osteomyelitis of dorsal vertebrae with abscess formation and stenosis of right upper lobe.

Fig. 15.—Case 11. Actinomycotic abscess of left base with contraction of left chest and elevation of left diaphragm.

a staphylococcic abscess of the eighth dorsal vertebral body with a large mediastinal abscess. At this time he had a distressing dry barking cough and occasionally spat up small amounts of blood. There was massive dullness with loud amphoric breathing over the hilum. A large mediastinal abscess containing two to three cupfuls of greenish pus was opened and drained through a posterior incision, after which the fever receded, the cough ceased and he gained weight and strength.

Actinomycosis may act in the same way. Serous mediastinal exudates and encapsulated mediastinal pleurisies and empyemas may compress and obstruct the trachea and bronchi.

CASE 11.—An Italian gardener of twenty-nine entered the San Francisco Hospital on September 20, 1927. Two and a half months before he had had a sudden pain

in the left chest and the next day spat up a teaspoonful of bright blood. Five weeks before entering he began to expectorate foul sputum. The left side of the chest was contracted and the left diaphragm raised. The left base was dull and the breath sounds over it were diminished. Operation revealed an encapsulated actinomycotic abscess lying in the gutter between the ribs and the vertebral column. Retroperitoneal abscesses appeared and the patient died April 21, 1928.

Bronchial stenosis from sclerotic peribronchitis and lymphadenitis in the aged has been well delineated by the pathologist Schmorl[27] under the name of *bronchitis deformans.*

*Intrinsic stenoses* are caused by foreign bodies, by tumors growing into the tracheobronchial lumen, by constricting intramural inflammatory processes and their scars, and by softenings of unknown origin. The pathology and clinical aspects of foreign bodies have already received full attention.

Tumors of the bronchi, innocent and malignant, especially carcinomas, which seem to be steadily increasing in frequency, are among the most common intrabronchial causes of obstruction. The stenosis occasioned by them may be intermittent at first as the tumor mass swells more or less with increase or decrease of concomitant inflammation or engorgement, but final closure is complete unless ulceration again opens the air passages.

Tracheal carcinoma, although rarer than either laryngeal or bronchial carcinoma, is not unknown. E. Fraenkel[28] has described ten cases of his own. An interesting variety of malignant and nonmalignant tracheal tumors are known to bronchoscopists as causes of compression. Thirty cases of small goiter nodules have been described, lying in the tracheal wall, quite separate from the rest of the thyroid gland. They represent aberrant congenital thyroid remnants; they may be attacked by cancerous changes.

A disease designated as chondro-osteoplastic tracheopathy, characterized by the formation of exostotic and ecchondrotic ridges seems to be not at all uncommon and known to both pathologists and laryngologists. The affection usually involves the trachea, rarely the larynx, occasionally the bronchi; it occurs more frequently in women; 25 per cent of all cases are tubercular. Its origin is obscure. Occasionally the growths may block the trachea or bronchi. A localized form of amyloid degeneration affecting larynx, trachea, and bronchi leads to the formation of granular and tubercular excrescences, yellowish and dark red, of colloidal consistency, which occasionally constrict the tracheal lumen. Finally leucemic lymphoblastomas occur in the wall of the trachea and at the bifurcation which may reach the size of a bean and cause obstruction. Cysts and diverticula of the tracheal wall seem to be very common. They are so similar in appearance to peritracheal lymph nodes that they are usually mistaken for them at autopsy, and are distinguishable from them only upon section. There has been much discussion concerning their etiology: whether they are to be regarded as hernias of the tracheal mucosa or retention cysts

caused by closure of the ducts of the tracheal mucous glands. Whatever their origin may be, erosion and ulceration of the cyst wall, with suppuration, caseous degeneration and also tuberculosis have been described, leading to irritation and ulceration of the trachea and narrowing of its lumen.[29, 30]

*Inflammatory stenoses* are no less varied.

Syphilitic scars constrict the upper air passages and sometimes the greater bronchi; they are more frequent in the larynx and trachea and at or near the tracheal bifurcation than lower down. They are the result of gummas and syphilitic infiltrates, proceeding with or without suppuration, causing bronchotracheal perichondritis, destroying the cartilages and leading to extensive scarring and distortion of the tracheobronchial tubes. Nonulcerative syphilitic intrapulmonary peribronchial sclerosis may also constrict the lower bronchi.[31, 32]

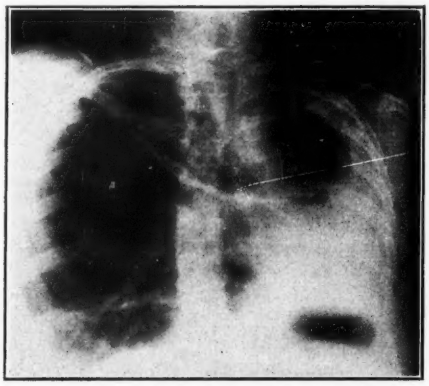


Fig. 16.—Case 12. Narrowing of tracheal lumen at its middle. Contraction of left chest with huge cavitation of upper lobe and atelectasis of lower lobe. Tuberculosis of the trachea.

Old tubercular submucous infiltrates occasionally produce thickening sufficient to obstruct respiration, a thickening which at first glance may be mistaken for a malignant one.

CASE 12.—A woman of thirty-seven, seen with Dr. P. K. Brown in 1920, had had a tuberculous pneumonia twelve years ago, and a year later "asthma," increasing with exertion. For the last three months she had had severe dyspnea, wheezing, stridor and strangling fits, mainly at night. The x-ray revealed an apparent interruption in the course of the trachea with a widening of the lumen below. The thoracic viscera were displaced markedly to the left. Bronchoscopy revealed a tracheal obstruction at the level of the second sternochondral junction. Mediastinotomy revealed no outward pressure, but that of the aorta which pushed the trachea to the right. The patient died. At autopsy the tracheal mucosa at the crossing of the innominate artery was found to be red, pitted and the tracheal wall so thick that the lumen of the trachea barely admitted a lead pencil. Dr. Ophüls reported a marked circular stenosis of the lower end of the trachea and dilatation above and below. The microscopic diagnosis read: Tuberculosis of the trachea.

Tubercular peribronchial sclerosis more often affects bronchi of the second order, as will be seen later, although it may also distort and constrict the major ones.

Rhinoscleroma and leprosy may lead to similar changes. Glanders usually makes ulcerative lesions like tubercular ones which if they heal may leave constricting scars. Typhoid may cause not only laryngeal but tracheal perichondritis with necrosis of the cartilage, tracheal deformity and stenosis.

Finally a tracheomalacia of unknown origin has been described which from its form would seem to be due primarily to changes either in the elastica or musculature of the posterior membranous portion of the trachea rather than in the cartilaginous rings. The anteroposterior diameter of the trachea is flattened, the transverse one lengthened; the posterior wall is elongated; the cartilages instead of their normal horseshoe form, assume the shape of a cupid's bow. It would appear that the tensile forces holding the cartilaginous horseshoe in shape have slackened and allowed the rings to spring out.

Disorders affecting the large bronchi usually affect but one side, either the right or left major bronchus, syphilis and rhinoscleroma excepted, which from the diffuseness of the scars and predilection for the region of the bifurcation often constrict both sides at once. Tumors ordinarily block but one lobe or even a part of one; they arise more frequently in the intrapulmonary parts of the bronchial tree than in the two major bronchi. Tubercular glands and nontubercular inflammatory constrictions also block but one of the larger bronchi as a rule; not infrequently (as is natural from the position of these glands) a major bronchus, obstructing a whole lung, less often both. Acute suppurative lymphadenitis of children is an exception; these glands often perforate the trachea itself.

Aneurysm of the descending aorta and the arch and dilatation of the left auricle block the upper left bronchus and leave the right side free; aneurysm of the ascending aorta usually obstructs the right side.

Stenosis of the middle bronchi, from those of the second or third order down, is due either to small foreign bodies or to diffuse sclerosing processes. Pneumoconiosis makes sclerotic peribronchial constrictions, so does tuberculosis, and most strikingly a combination of the two, both of them diffuse, affecting the smaller bronchi of the third and fourth order, extending occasionally to the bronchioli. Distal to them one finds the alveoli blown out into large emphysematous bubbles. The "asthma" of the tuberculous I suspect to be more often due to this cause than to a truly asthmatic hypersensitiveness.

CASE 13.—A case in point is that of Mr. B., a man of sixty who had worked as a quartz miner for twenty years. He quit the mines in 1922 on account of cough. He had coughed for the last six or seven years, perhaps longer, bringing up a greyish and yellowish sputum, streaked with small flecks of blood. He had never had a severe hemorrhage. On examination in May, 1927, he was thin and ex-

ceedingly dyspneic, wheezing when he breathed. The right base was dull, breath sounds were diminished over the right. Coarse dry râles were scattered diffusely over both sides of the chest, with coarse moist râles over the right apex anteriorly. The veins of the upper chest were moderately dilated. The rib cage was rigid, but both sides moved equally. The blood count was normal. No tubercle bacilli were to be found on repeated examination. He denied venereal infection. The Wassermann reaction was once negative, once doubtful.

Fig. 17. Fig. 18.

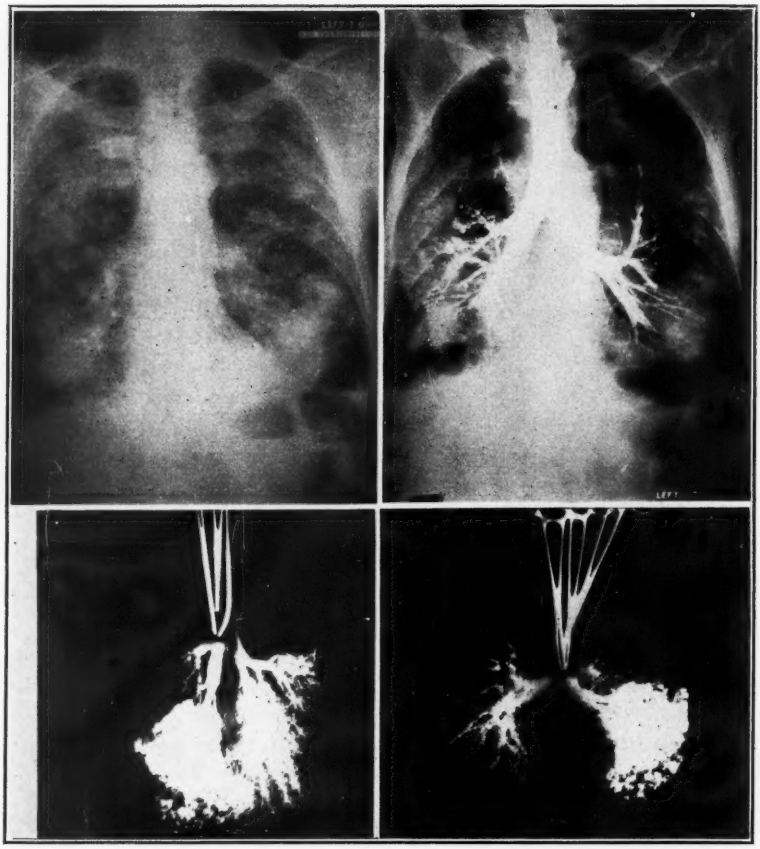



Fig. 19. Fig. 20.

Fig. 17.—Case 13. Central pneumoconiotic deposits; peripheral emphysema most marked at bases; diffuse stenosis of small bronchi.

Fig. 18.—Case 13. Postmortem specimen; barium gelatin injection fills larger bronchi only. Smaller bronchi not injected.

Fig. 19.—For comparison. Normal lung injected with barium gelatin; the finer bronchi are filled on one side, the alveoli on the other.

Fig. 20.—For comparison. Normal lung injected with barium gelatin; the finer bronchi are filled on one side, the alveoli on the other.

Dr. Rehfisch reported the x-rays as follows:

"Chest: The subclavicular portion of both lung fields is diffusely sown with dense cottony areas of mottling which become more extensive and opaque as one

approaches the two bases. The apices are relatively clear. The mediastinal contents are pulled somewhat to the right and the lateral view shows a practically opaque posterior mediastinum. The diaphragmatic outline is most irregular on both sides, due apparently to extensive pleural diaphragmatic adhesions.

"Fluoroscopically the diaphragms show practically no motion on either side.

"Conclusion: Advanced pneumoconiosis."

The patient died during a suffocative attack. *Autopsy* revealed enormously dilated lungs, the left one reaching into the right chest to the right sternal border and overlying the heart. The diaphragm lay at the level of the sixth rib.

There were perhaps two ounces of clear, yellowish fluid in the heart sac. The heart was a little larger than the fist; it contained large quantities of still liquid dark blood. The right ventricle was somewhat dilated and its wall thickened to perhaps ⅜ inch. There were some slight patches of atheroma around the mouth of one coronary, but the aorta and coronaries were remarkably soft for a man of his years.

Both lungs were attached to the chest wall by numerous bands of firm adhesions. The pleural cavities were by no means obliterated; there was no free fluid in them. The lungs were so greatly dilated that they could be with difficulty extracted from the chest.

In both lungs, especially the left, were numberless bubbles of emphysema, one of them the size of the palm of the hand and occupying the apex of the left upper lobe; there were numerous others the size of a small pullet's egg, making both lower lobes appear knobby and lobular. The walls of these sacs were thin as paper.

In the center of both lungs one could feel a mass of chalky hardness threading its way through the parenchyma, evidently following the bronchi. In the right lung the surface section of this central hardness was about the size of a dollar.

The lungs were purple and much congested, as were all of the inner viscera—both thoracic and abdominal. Along the hilum and trachea lay packets of firm black glands, the largest the size of a walnut. These did not appear at any place to constrict the tracheal or bronchial lumen and I think that the constriction lay at the level of the bronchi of the second order or lower.

The liver lay four fingers below the ribs in the right axillary line; it was mottled and congested. The spleen was small, fibrous and firm. The other viscera were markedly congested, but otherwise normal.

Barium injected into the cadaveric trachea filled the bronchi of the first and second order and then stopped; the finer bronchi and bronchioli were not injected.

The following instance of tubercular asthma, so called, was also probably dependent on a diffuse stenosis of the smaller bronchi, together with a stenosis of the left major one.

CASE 14.—Mrs. R. K., a woman of twenty-nine had two children of eight and five years. An uncle and a sister were asthmatic. After the birth of the second baby she had an illness thought to be influenza and two months later had her first attack of asthma. These attacks have continued since; they last six weeks and are so severe that she is confined to bed and has to sleep sitting up. They are accompanied by much coughing and are due to no known cause. They are ushered in by attacks of sneezing. Her temperature is usually normal.

On examination June 30, 1927, the sternal notch and the supraclavicular fossae were retracted on inspiration, but expiration was free. The chest was flat rather than barrel-shaped. Purring and snoring ronchi were heard everywhere over both lungs. The breath sounds were much diminished especially at the base where they were scarcely audible. At about the level of the scapula the breath sounds began

to be normal on both sides. The heart was not enlarged; the sounds were ringing, as though the heart were working under difficulties.

The red cell count was 4,800,000; hemoglobin 88, the white cells were 16,200. After much search tubercle bacilli were demonstrated in the sputum by the antiformin method. Skin tests with a hundred different foreign proteins gave no positive reaction. Dr. Rehfisch made the following x-ray report:

"Chest: Fluoroscopically both lung fields give the impression of markedly increased translucence. The diaphragm does not move at all on either side and both domes are deformed by puckered pleural adhesions.

"The plates show pleural scarring at both apices and between the lobes on the right. There are definite mottled shadows at both tops and the peribronchial markings present an unusually beaded appearance throughout both lung fields. Whether this is entirely a question of artefact due to the increased amount of air in the emphysematous lungs or whether the beaded shadows represent actual foci

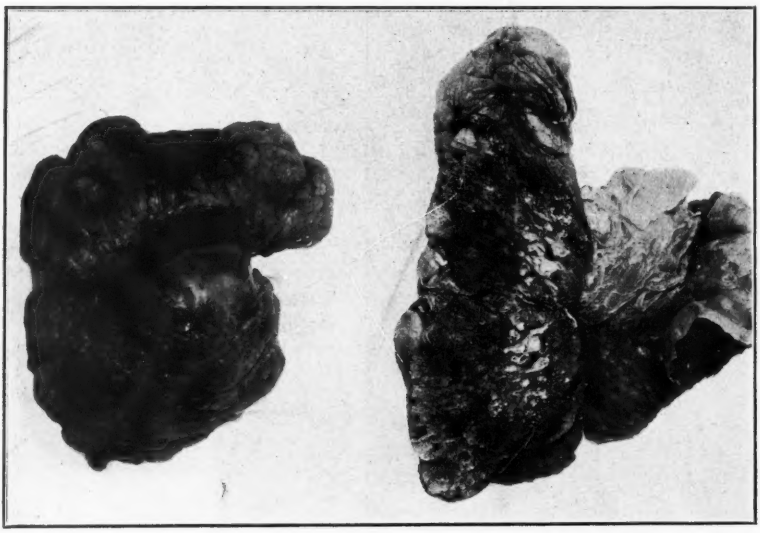


Fig. 21. Fig. 22.

Fig. 21.—Case 13. Dilated lungs viewed from above, showing large emphysema bubbles.

Fig. 22.—Case 13. Section of lung showing central fibrosis. Small emphysematous vesicles in the subcortical area; large bullae of emphysema, one of which is cut open.

of consolidative pathology is difficult to say. That such pulmonary pathology exists at the apices at least, however, is extremely probable.

"Conclusion: Emphysema. Pleural adhesions and thickening. Apical pulmonary tuberculosis with probable acute disseminated tuberculosis of the miliary type."

Dr. Harold Fletcher did a bronchoscopy on August 14, 1927, and reported as follows:

"Examination showed nothing abnormal in the throat, hypophyarynx or trachea, except a slight suspicion of flattening along the right wall of the trachea. The bifurcation was normal; the right bronchus was somewhat congested and had a very slight tendency to bulge in from the left side about 1 to 1½ inches below the bifurcation. Also, the direction of the right main bronchus tended to incline more to the right than usual. The left main bronchus was almost completely closed

about ½ to ¾ inch below the bifurcation. This closure appeared to come from a bulge in the wall from the left and somewhat anteriorly. The remains of the lumen gave the appearance of being flattened out even from the right posteriorly.

"This examination checked your preliminary diagnosis of stenosis of the left main bronchus."

At several subsequent examinations the breath sounds were consistently diminished on the left side. She was discharged to her home in Napa, where she died in a suffocative attack. She was very blue and her doctor said her heart had given out.

Sergent[32] and Benda[31] have drawn attention to syphilitic peribronchitis, which causes dense sclerosing infiltrates, at times obliterating and obstructing the bronchial lumen.

*Constriction of the bronchioles* and obstruction of their lumen by swelling and destruction of the mucosa follow inhalation of destructive fumes and gases, but also occur more or less extensively after infectious bronchitides, influenzal infections, and after the syphilitic processes mentioned above. The changes were first noted by Lange[33] and were afterward described by Fraenkel[34] under the term "bronchiolitis fibrinosa." They are among the later sequels of inhalation of war gases. Obstruction of the bronchioles may also be, if not the cause at least the accompaniment of certain congenital atelectases.

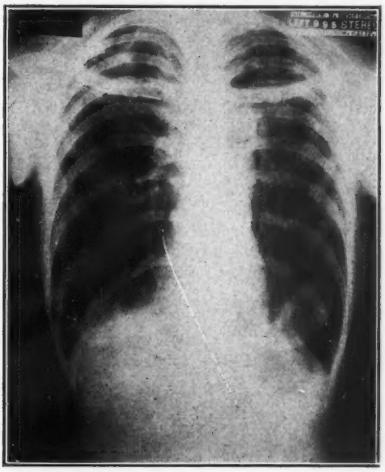


Fig. 23.—Case 14. Diffuse tubercular stenosis of smaller bronchi. Marked dilatation of lungs and rib cage. Note wide chest and narrow waist. Small heart.

Many of these causes of obstruction lead to similar results. The results vary with the site of stenosis, its degree, its duration and with complicating factors, among which chronic and acute infections play a decisive rôle. Given the site and degree of an obstruction it should be possible to deduce its effects, and vice versa, to locate its site given its effects.

*(To be continued. Bibliography to follow Section III.)*

# Department of Reviews and Abstracts

Conducted by Duff S. Allen, M.D., Associate Editor

## Selected Abstracts*

### Physiology

**Romankevic, V.: Topographische-Anatomische untersuchungen uber den Lungenabschnitt des Vagus und die Bronchialgeflechte. (Anatomical Study of the Pulmonary Divisions of the Vagus and the Bronchial Plexus.)** Deutsche Ztschr. f. Chir. 231: 8-9, July, 1931.

The surgical treatment of bronchial asthma has assumed importance, but probably because of the lack of an exact and complete knowledge of the anatomy of the innervation of the lungs, has resulted in a great variance and confusion of interpretations and results. Studies were undertaken to ascertain the exact topographical anatomy of the pulmonary innervation and to determine the most feasible surgical approach. The description of the anatomy, as detailed, is taken from the dissections of fifty cadavers. The studies on the operative approach were carried out on ninety cadavers. The following conclusions are drawn:

1. The division of the vagus trunk usually begins in the uppermost portion of the bronchi.

2. The number of anterior bronchial branches usually is three to four—may be as high as six or as few as two.

3. The anterior bronchial nerves branch off from the vagus trunk in the region between the recurrent laryngeal nerve and the upper border of the bronchus.

4. In fully 50 per cent of the specimens, combined branches were found situated well anteriorly from which the posterior branches sprung.

5. A portion of the left anterior bronchial nerves and the tracheal branches always receive branches from the right vagus for innervation of the left bronchi and the left lung.

6. The number of posterior bronchial nerves varies from five to six to ten to twelve.

7. The division of the vagus trunk from which the bronchial nerves arise is larger on the right than on the left. The upper border of the branching of the posterior bronchial nerves is deeper on the left than on the right.

8. Two types of structure of the posterior bronchial nerve plexi were noted—a large compact unit and a fine scattered arrangement. The former is characteristic for the left side, the latter is characteristic for the right side.

9. The surgical approach for the bronchial nerves on the right must be through a very large wide exposure whereas that for the left nerves can be performed through a smaller but deeper exposure.

*The abstracts in this issue were compiled by Dr. M. M. Berck, Research Fellow in Thoracic Surgery, Washington University School of Medicine, St. Louis.

10. In some cases a blending and admixture of the mediastinal branches of the sympathetic with the anterior bronchial nerve branches of the vagus was clearly demonstrated.

11. The theoretical possibility of complete denervation of the lung would seem to be soon practical through the operation of von Sudeck—resection of the anterior and posterior bronchial nerves.

12. The anterior transpleural approach to the nerve structures at the hilum is thoroughly inadvisable.

13. The posterior transpleural approach is likewise to be avoided because of the pleural adhesions present in certain cases which prevent exploration and obscure the landmarks.

14. The posterior extrapleural route exhibits the possibility of displaying the entire vagus trunk throughout its length with the anterior and posterior branchings of the bronchial nerves.

15. A three-stage extrapleural procedure is advisable to avoid damage to the pleura—employing a technic similar to that used in extirpation of the esophagus.

**Dolainskie, A.: Ueber Sympathectomie bei Asthama Bronchiale. (Sympathectomy for Bronchial Asthma.)** Mitt. a. d. Grenzgeb. d. Med. u. Chir. 3: 1931.

The reports on the experiences with sympathectomy in bronchial asthma are so various and the entire sphere of asthma investigation is so full of contradictions and highly unclassified that the analysis of operated cases permits no outstanding conclusions. Moreover, the operating technic is very recent and still in the process of development. It is very probable that none of the proposed operative technics afforded an exposure of sufficient extent to enable one to completely extirpate either the vagus or the sympathetic. It is to this fact alone that many failures can be ascribed. A knowledge of the exact point of surgical attack—vagus or sympathetic —is by no means settled. Many investigators cannot be convinced from their observations that the vagus alone carries the bronchoconstrictor fibers. The consequence has been that operations have been performed on both the vagus and sympathetic. A statistical summary shows that with sympathectomy at all events there is the high proportion of 47.5 per cent of failures. This great number of failures can be partially ascribed to the expectations of the patients who await an immediate curative result after the performance of the operation. It is necessary to make it understood that recovery from the concomitant symptoms and complications of the asthma; namely, emphysema and bronchitis, takes place only over a long period of time and that the operation, by removing the etiologic factor (though this is unsettled) cannot affect the complications of the disease.

The point of greatest importance is to be able to completely sever the innervation of the lung. Further surgical research must be undertaken to develop a feasible technic for this. From the report sympathectomy for bronchial asthma is still to be regarded as a very uncertain measure, but because of its relative safety, its possibilities and the results obtained with isolated cases, it is not to be abandoned.

**Powers, J. H., and Pilcher, C., and Bowie, M.: Some Observations on the Circulation in Experimental Mitral Stenosis.** Am. J. Physiol. 97: 405, June, 1931.

The present consensus of opinion concerning heart disease in general favors the premise that the cardiac output per minute is decreased in proportion to the degree of cardiac damage. These experimental results do not support this conten-

tion. The operative technic and subsequent bacterial injections essential to the experimental production of mitral stenosis have been previously described by Powers; viz., the heart is exposed through a subperiosteal resection of a portion of the fifth rib, the active electrode (diathermy producing a bipolar current) is approximated to the mitral ring and inferior surface of the leaflets of the valve and the current is applied several times for one to three seconds. During the early postoperative period each animal is inoculated intravenously with fresh broth cultures of Streptococcus viridans. Each dog developed accentuated cardiac sounds, a systolic murmur, and showed a positive blood culture for several weeks. As the lesions healed, organisms disappeared from the blood stream and the operation was repeated. At the second operation some degree of stenosis of the mitral orifice and multiple scars around the opening were palpable in every case. Each animal was then reinoculated with larger quantities of streptococci than before. All developed a pronounced bacteremia presumably due to a fresh superimposed vegetative lesion on the mitral valve. Presumably because of the resistance developed in overcoming the initial infection, organisms disappeared from the blood stream relatively early as compared with the period after the first operation. At intervals of three to ten months later, observations were carried out. The studies on a control animal that was subjected to all the operative features save the electrocoagulation of the valves showed no variations from the normal.

Observations were made on some phases of the circulation of five dogs with experimental mitral stenosis. The cardiac output of four of the animals was within the range of normal. That of a fifth animal was distinctly elevated. The basal pulse rate was increased in four instances and normal in the fifth. The blood pressure was elevated in only one instance. An increase in the red blood cells occurred at the expense of the plasma. The size of the heart as determined by x-ray showed a progressive enlargement to the right in all five dogs. No significant changes were observed in the EKG tracings.

**Beard, J. W., Butler, V., and Blalock, A.: A Study of the Division of the Cervical Esophagus of the Dog.** Surg., Gynec., and Obst. V. 53: 169, August, 1931.

Complete obstruction of the esophagus of the dog produces death in one to three days. Studies on the blood after occlusion of the esophagus showed that there is usually an increase in the nonprotein nitrogen and urea nitrogen and a decrease in the chloride content. These alterations were not sufficiently constant or great to indicate that they were responsible for the early death. Dogs with fistulas of the esophagus die in almost as short a time as those in which the esophagus is simply obstructed, suggesting that the loss of fluid causes the death after occlusion of the esophagus. If the loss of fluid as saliva is responsible for the death of the animals after the esophagus is obstructed, a concentration of the blood, a decrease in the blood volume and a decrease in the content of water in the tissue of the body would be expected.

The blood volume, the plasma volume, the red blood cell count, the hemoglobin, the chloride content of the blood and the content in water of blood and muscle were determined before and after: (1) division of the cervical esophagus; (2) food and water deprivation is eighty hours; (3) graded removal of blood plasma over a period during which the animals were not allowed food and water; (4) graded removal of whole blood during a period of water and food deprivation.

The alterations which were produced by ligation of the esophagus and by removal of the plasma were quite similar. The findings suggest that dehydration which is probably in the main due to the loss of saliva is largely responsible for the early death that follows division of the esophagus.

**Polland, W. S., and Blomfield, A. L.: Experimental Referred Pain From the Gastrointestinal Tract. Part I. Esophagus.** J. Clin. Investigation, 10: 453, August, 1931.

Pain was produced by inflating balloons with air at various levels of the esophagus. The following points were studied: (a) The minimum amount of inflation necessary to produce sensation and (b) the site of the sensation, (c) the character of the sensation, (d) the relation of the site of sensation to the position of the balloon and (e) the relation of sensation to spontaneous clinical symptoms. A miscellaneous group of patients free of gastrointestinal trouble was examined.

It was found that the site of the referred pain bears no relation to the position of the balloon but occurs especially at the xyphoid or in the suprasternal notch and less often over the anterior chest wall or in the back. Rarely it was felt in the neck or face and in two cases nausea was produced. This is in variance with those reports which have stated that pain from distention of the esophagus is referred to the body surface (sternum) at the level of the site of distention.

The character of the sensation was crude and often could not be accurately described or delineated by the patient.

No relation could be made out between the subject's general susceptibility to pain and the ease with which referred pain was produced.

## Mediastinum (Neoplasm)

**Lamarque, P., and Bert, A.: Tumeurs de Mediastinum et Radiotherapie. (Mediastinal Tumors and Radiotherapy.)** Arch. Soc. d. sc. méd. p. 186, March, 1931.

Two lymphoid mediastinal tumors have been observed to recede under the influence of intensive radiotherapy. A review is made of the sensitivity of different mediastinal tumors to radiation. The two cases were:

1. A child of fourteen years with a lymphocytoma (biopsy) of the mediastinum which was treated with intensive radiotherapy and now appears clinically cured with complete disappearance of the mass and great increase in weight and strength.

2. The other case is that of a lymphogranuloma (biopsy) in a male of twenty-seven. The tumor completely disappeared within a year under intensive radiotherapy with great gain in weight and return to occupation. More recently the patient has contracted pulmonary tuberculosis and is receiving pneumothorax.

The symptomatic unity of mediastinal tumors conceals the differentation of a number of types. A therapeutic test of radiation will serve to differentiate them. The authors quote the work of Evans and Leucutia in this regard. The application of a definite amount of radiation will alter the size of certain mediastinal tumors within determined times. According to this, one may classify mediastinal tumors into four groups:

1. Complete disappearance of the tumor in four to ten days of radiation. These are tumors of the lymphoid cells in the mediastinal nodes or the thymus, i. e., lymphoma, lymphosarcoma, lymphatic leucemia, thymoma.

2. Partial reduction in size in ten days, complete disappearance in six weeks of radiation. Tumors of the reticuloendothelial cells of the same structures (Hodgkin's disease).

3. Slight or moderate reduction in size without disappearance in six weeks of radiation; teratoma, round-cell sarcoma, thymic carcinoma.

4. No alterations in size. Benign tumors and pseudotumors.

The technic employed consists in massive radiation with a dose of 90 to 100 per cent of the standard skin dose, with the penetrating rays of 200 KV without filtration.

Lymphoid and hemopoetic tissues possess a remarkable sensitivity to intensive radiation. This has been shown to be dependent upon massive necrotic changes of these cellular elements which is followed by a great infiltration of polynuclear leucocytes which phagocytize the cellular débris. The combined action of these two mechanisms explains the selective and often prolonged reactions to radiation of these tumors and enables one also to comprehend the rapidity with which some extremely large tumors seem to melt away. Although often of only temporary efficacy, radiotherapy is the most potent weapon in the curative or palliative treatment of these mediastinal tumors.

## Trachea, Lungs, Pleura

**Matas, R.: On the Present Status of the Postoperative Pneumopathies.** International Clinics. II. 41. p. 1, 1931.

The statistical study of the postoperative pulmonary complications has been deficient and defective because of lack of agreement on nomenclature, the failure to record milder complications and the variety of interpretations given to common pathologic states. The pneumopathies is a term used in the generic sense to embrace a variety of pathologic states or lesions in which infection, embolism and atelectasis are the underlying causative factors. This paper is confined chiefly to the acute complications which occur during the first five days or week following operation. A classification of them is: (1) bronchitis and bronchorrhea. (2) Pulmonary congestion. (3) Lobular or bronchopneumonia. (4) Lobar pneumonia. (5) Embolic pulmonary obstructions leading to infarctions and pleuropneumonia. (6) Pleurisy. (7) Atelectasis.

The risk or liability of the patient operated upon to postoperative complications has been variously estimated to from 0.5 per cent morbidity with 0.3 of 1 per cent mortality, to 4 per cent morbidity with 3.3 per cent mortality. The relative immunity of children to postoperative pneumopathies is significant as is the rarity of postoperative pulmonary embolism in childhood. The greater liability of operations in the upper abdominal belt to pulmonary complications is estimated as ten times as great as the incidence after lower belt laparotomies. The theories that are uppermost in accounting for the pathogenesis of pneumopathies are:

I. The Inhalation Anesthetics. (a) The direct irritant and toxic effect on the bronchi and lung parenchyma; (b) aspiration of septic material from the mouth and nasopharynx or the entrance of vomited matter into the larynx in the course of general narcosis. It cannot be denied that anesthesia cannot be altogether eliminated from the etiology of the postoperative pulmonary complications.

II. The Specific Air-borne Infections. (a) The objections that have been advanced against pneumococcal aspiration infections as the dominant cause of the postoperative pneuminitis are:

(1) It is admitted by all investigators that bronchial aspiration occurs more or less constantly under general anesthesia, as in tonsillectomies, but that postoperative pneumonias rarely result therefrom. (2) That the experimental pneumonia work does not reveal much success with inhalation infection except with extremely large doses not comparable to clinical inhalations. (3) That the pneumococcus IV is found in the mouths and sputum of normal individuals and its presence in the expectoration is no sign of its specificity. (4) That x-ray and clinical findings might also be interpreted in the light of embolic infarcts or atelectatic patches. (5) That the postoperative pneumonias continue to appear

in patients operated upon by local and regional anesthesia despite the total exclusion of inhalation anesthetics.

III. Endogenous Infections: Infection may be carried from the field of operation in the abdomen to the lungs by three possible routes: (1) Porto-caval anastomoses. (2) By the veins of the abdominal wall. (3) By the abdominal lymphatics emptying into the thoracic ducts and thence into the innominate vein. A very considerable mass of experimental evidence has accumulated in recent years which fully confirms this possibility.

IV. Embolic Theory: This involves two categories of emboli: (a) Minute hypothetical emboli which reach the lung from the field of operation through venous and lymph channels. (b) Those emboli appearing later, in the second week, which are larger and may become massive, obstructive and fatal. The chief objection to the theory of embolic showers from a clean abdominal incision is that it is entirely a hypothetical assumption incapable of demonstration and contrary to our knowledge of the behavior of hemostatic wound thrombosis in clean abdominal incisions. The infarctions, due to minor and major emboli do not occur until the beginning of the second week.

V. The Atelectatic Theory: The theory has been advanced of the unity of all the postoperative pneumonias, that they are dependent essentially upon one cause, atelectasis. According to this view embolism and infection are minor or secondary in the causation of a pneumonic state, atelectasis being always the first stage. The theories concerning the etiology of atelectasis are two: (1) The absorption theory of alveolar collapse (atelectasis secondary to mechanical mucous plug of the bronchi). (2) Expulsion of the alveolar air by bronchopulmonary spasm. In connection with the second theory the recent work of Luisada is interesting. This author has shown that the whole lung is a muscular organ, that not only the bronchial muscle contracts but that the lung itself participates in the contractions and the air in the alveoli is forced out coincidentally with the bronchial spasm. A partial atelectasis is thus produced and is completed when the walls of the terminal bronchiales are brought into apposition. The discovery of the electrobronchograph has shown that the bronchopulmonary muscle has automatic, rhythmic movements of its own, entirely independent of the external respiratory movements or other extrinsic influences. The lung has regular rhythmic movements of contraction and relaxation which occur synchronously with the respiratory movements and yet are independent of them. The contractions are stimulated into activity by the distension of the bronchi and are most strikingly manifested in the phase of spontaneous retraction which follows bronchial distension. In the researches using the electrobronchograph the following have been noted:

(1) An abdominopulmonary reflex is roused by mechanical stimulation of the stomach and the solar plexus which induces a reduced ventilation of the lung and a diminution in the expulsive power of the bronchi. This fact contributes to a retention of the bronchial secretions and to an alveolar atelectasis, conditions which are recognized as the most important factors in the pathogenesis of collapse and postoperative pneumonitis.

(2) The fact that blocking the abdominal sympathetic with a local anesthetic suppresses the abdominopulmonary reflex would seem to account for the relative freedom from pulmonary complications that is claimed by many surgeons for splanchnic anesthesia and high spinal, when used for laparotomies. The marked influence upon the musculature of the lung would seem to confirm the opinion that injuries of the solar plexus have some influence in favoring the development of the postoperative pulmonary complications.

(3) The depressing effect of ether and chloroform, when administered by inhalation, upon the contractility of the bronchopulmonary muscle, favors the stagnation and retention of bronchial secretions as well as a diminution in the alveolar aeration, which are most important factors in the pathogenesis of the postoperative pulmonary complications.

(4) Paradoxic as it may seem, the bronchograph demonstrates that morphia and ether (not by inhalation) in small doses have a marked stimulating effect on the musculature and mobility of the lung, which suggests the use of these drugs (contrary to our preconceived ideas) as prophylactic and therapeutic activators of the respiration in the postoperative pulmonary complications. All the evidence thus far gathered points to the possibility of causing bronchopulmonary spasm sufficient to expel the air from the alveoli and thereby produce atelectatic phenomena.

Some observers have reported that every case of postoperative atelectasis was definitely allergic in various ways. The individual constitution in susceptibility to allergic reactions may come into play to explain the occurrence in some individuals and not in others under seemingly identical conditions.

The attempt to explain all the postoperative pneumopathies on one basis and expect to unite these under one single etiologic factor is illogical and futile. The prophylactic treatment by inhalations of carbon-dioxide and oxygen immediately after operation promises to be very valuable.

**Felix, W.: Ueber die Plombierung in der chirurgischen Behandlung der Lungentuberkulose. (Plombage in the Surgical Treatment of Tuberculosis.)** Ztschr. Tuberk. **59**: 385, 1931.

The report is that of the practice of plombage in one hundred cases during the past ten years at Sauerbruch's Clinic. If allowance is made for the possibilities of complications, the method has been found quite feasible for many cases under the correct indications. Disturbances in the healing of the implanted foreign body plombe are manifest as exudates consequent to an aseptic foreign body inflammatory reaction or as suppurations in the plombage bed. Strict operative asepsis being taken for granted, an infection in the plombage bed can originate only from the pulmonary focus by lymph channels or through a perforation of the wall of a cavity. Removal of the plombe in cases of infection of the plombage bed should be delayed as long as possible or perhaps not done at all. In contrast to this in cases of perforation of a cavity one should remove the foreign body plombe at the earliest possible moment. Perforation of a cavity wall presents a distinctly different clinical picture from lymphatic infection of the plombage bed by reason of the greater violence of its local inflammatory reaction and by the presence of emphysema in the tissues surrounding the plombe. Likewise a distinction is to be made between acute perforation of cavities and late perforation of cavities in that the late perforation of cavities is not nearly so dangerous and does not demand such immediate surgical relief. In those cases in which the weight of the plombe causes mechanical difficulties, surgical measures are necessary to relieve the pressure. This is of particularly great importance in those cases in which the pressure is being exerted upon the right heart wall, particularly the right auricle with threatening dangerous disturbances of rhythm. The risks of aspiration infections are considerably smaller with plombe than with more extensive collapsotherapy. Occasionally a rigid cavity wall does not yield to the plombe pressure, plombage then may throttle by compression the efferent bronchus of the cavity with stagnation of the secretions and consequent untoward deleterious results.

To establish the correct indications is of primary importance:

1. Plombage is never to be used if pneumothorax is feasible or thoracoplasty can promise success.

2. If a partial pneumothorax is present over the lower lobe paraffin plombe over the upper lobe is inadvisable because of the possible danger of the descent of the plombage into the pneumothorax pocket. Occasionally plombage can be very advantageous as a complementary procedure in cases in which thoracoplasty has produced an unsatisfactory collapse of cavities. The primary indication for plombage is large upper lobe cavities.

During the past year frequent instances of obliteration of cavities following diaphragmatic paralysis alone have been noted. Considerable information has been obtained to clarify the indications to determine which cases phrenicexeresis alone can produce success. Further analysis of these cases will enable one to determine the type of case suitable either for phrenicexeresis or plombage.

In spite of numerous failures encountered in the review of the cases, the author feels that the procedure should have a further opportunity. Stress is to be particularly laid upon scrupulous care in operative indications and upon improvement in the choice of plombage materials and operative technic.

**Berard, M.: Apicolyse et Plombage. (Results of Apicolysis With Plombage by the Anterior Route.)** Lyon Chir. **28:** 4, July, 1931, p. 500.

The author presents four cases upon whom the author's method of anterior apicolysis with plombage was performed. These cases are too recently operated to permit of an appraisal of the value of the anterior route method—it being only nine months since the first case was operated upon.

However, in forty operations performed up to date there have been no serious operative accidents except in two cases presenting enormous apical cavities. In both cases a thoracoplasty was initially proposed to the patients but was refused In spite of his better judgment the author was induced to perform an apicolysis with plombage. He did not think these cases suitable for apicolysis because of the great size of the cavities and the poor nourishment and debility of the patients, reflected in the frailty of the thoracic wall. In both cases, within eight to ten hours the temperature rose considerably and signs of suppuration appeared in the wound with extrusion of the plombage. The cavity had ulcerated through and was discharging its contents into the operative field.

In spite of removal of the plombe and in one of the cases the performance of a complementary upper thoracoplasty of the first seven ribs in an attempt to efface the large extrapleural space and obliterate the cavity, the septic phenomena continued and both patients went on to die in two to four months.

These cases definitely confirmed the author in his opinion that apicolysis with plombage should be distinctly restricted to ulcerocaseous lesions or cavities clearly limited to the apex or the parahilar region and not extending below the third rib anteriorly and of dimensions no larger than those of a pigeon's egg. In lesions more extensive than these the manipulations directly in contact with the pleura are too dangerously delicate. It is far preferable in these cases to perform an upper thoracoplasty prefaced by a phenicectomy.

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